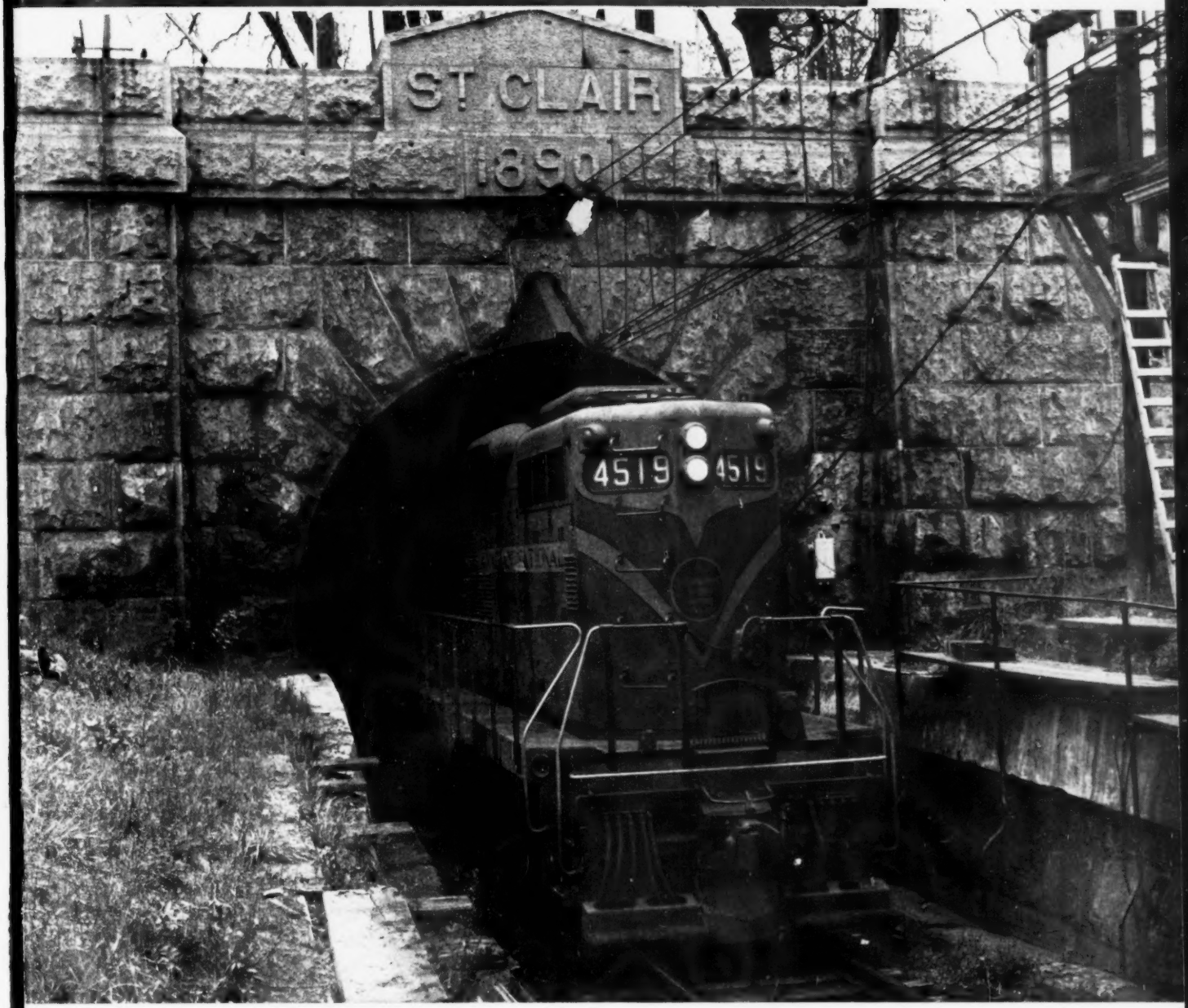
# OCCUPATIONAL HEALTH *Review*

*A Quarterly Publication*

VOLUME 11
NUMBER 3
1960

TON PUBLIC LIBRARY
APR 11 1960




*Occupational Health Division*

DEPARTMENT OF NATIONAL HEALTH AND WELFARE, OTTAWA

# Occupational Health Review

Volume 11, Number 3, 1960

The Occupational Health Review is published quarterly by the Occupational Health Division, Department of National Health and Welfare, with the co-operation of the Department's Information Services and under the authority of the Minister, the Hon. J. Waldo Monteith.

Chief, Occupational Health Division, T. H. Patterson, M.D., C.M., D.P.H., M.P.H.

Technical Editor, Irene Kingston, B.A.

Staff Advisory Board:

T. H. Patterson, M.D., C.M., D.P.H., M.P.H.

Mildred I. Walker, R.N., M.A., Senior Nursing Consultant.

Kingsley Kay, M.A., Ph.D., Senior Scientific Consultant.

Morris Katz, B.Sc., M.Sc., Ph.D., Officer in Charge, Environmental Unit and Air Pollution Consultant.

A. J. deVilliers, M.R.C.S., L.R.C.P., D.I.H., Officer in Charge, Biological Unit and Clinical Consultant.

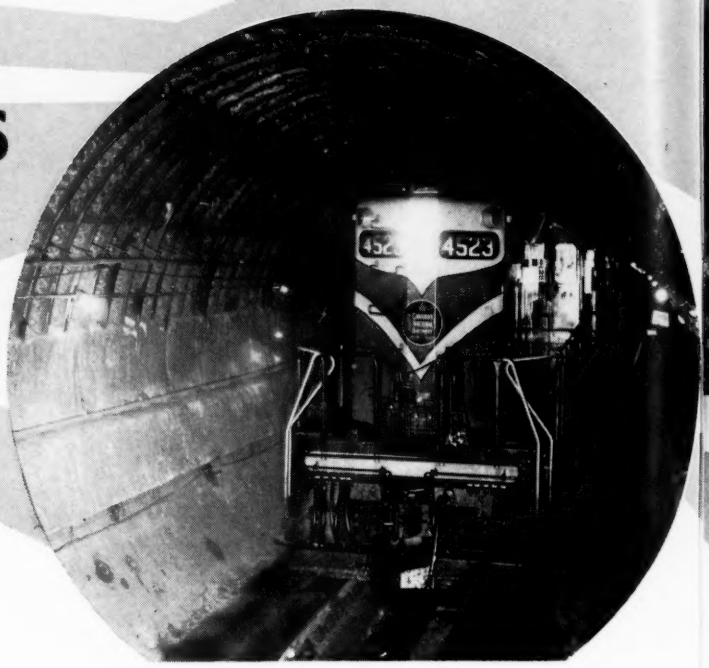
## TABLE OF CONTENTS

AIR POLLUTION AND ASSOCIATED HEALTH HAZARDS FROM DIESEL LOCOMOTIVE TRAFFIC IN A RAILWAY TUNNEL ..... Dr. Morris Katz ..... 2

HUMAN HEALTH AND THE MODERN ENVIRONMENT ..... Dr. Robert A. Kehoe ..... 16

THE TOXICITY OF TRICHLOROETHYLENE ..... Dr. Jonathan W. Williams ..... 22

# AIR POLLUTION AND ASSOCIATED HEALTH HAZARDS FROM DIESEL LOCOMOTIVE TRAFFIC IN A RAILWAY TUNNEL

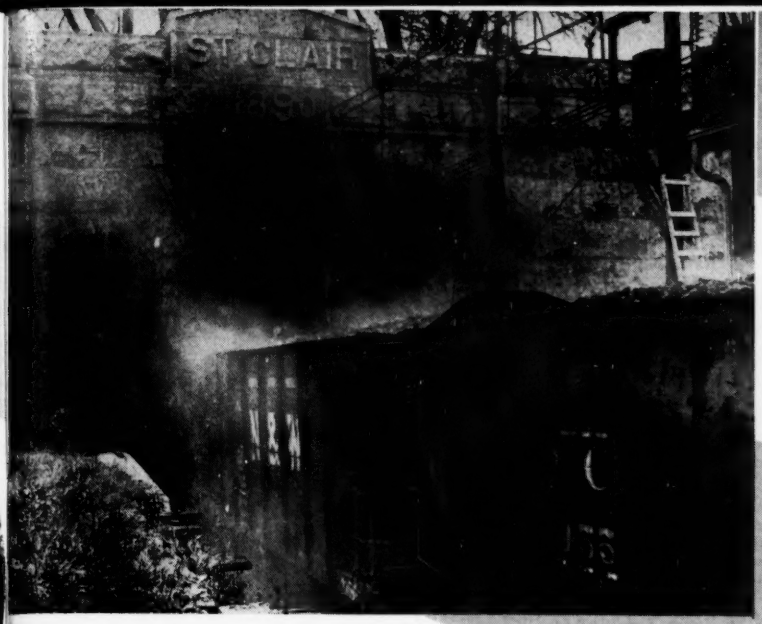



## INTRODUCTION

During October, 1957, an intensive study was made of the extent of air contamination from the exhaust gas of 1750 H.P. Diesel Locomotives (General Motors Class GR-17 Road Switcher) while hauling freight and passenger trains through the St. Clair Tunnel of the Canadian National Railways, linking Sarnia, Ontario, with Port Huron, Michigan. At this time the tunnel railway system was under electrified operation but plans for the dieselization of this section of the line raised the problem of the extent of air pollution to be anticipated from diesel exhaust fumes in the tunnel. The purpose of this survey was to determine the concentration levels of smoke, particulate matter and principal gaseous contaminants from diesel trains operated in accordance with the normal freight and passenger traffic schedules; to determine the rate and pattern of air flow through the tunnel induced by the train movement, to assess the possible health hazards due to the tunnel atmosphere and to evaluate the ventilation requirements for safe environmental conditions.

Extensive studies have been carried out by the U.S. Bureau of Mines on the use of relatively low horsepower diesel mine locomotives in tunneling operations underground. Ash and Naus[1] have reviewed some of the hazards involved in such diesel operations and concluded that the most toxic components of the gaseous pollutants in the exhaust are the oxides of nitrogen. However, there has been little published information on the exhaust of high horsepower diesel engines or on atmospheric conditions in railway tunnels. In 1946, Berger and McGuire[2] reported the results of a study of the composition of exhaust gas and resultant air comtamination in a railway tunnel from a 4-unit diesel locomotive, of which each engine developed 1570 H.P. at maximum speed. Their observations on atmospheric conditions in the Cascade Tunnel, following various passages of a train equipped with the above diesel locomotive, indicated maximum levels by volume of 0.53 percent $CO_2$, 0.014 percent carbon monoxide and 171 p.p.m. of oxides of nitrogen (calculated as $NO_2$) in the six samples analyzed.

This present paper describes the results of an intensive study of a railway tunnel environment under conditions of fully dieselized train traffic. Atmospheric sampling tests were conducted around the clock on an 8-hour working shift basis during October 18, 22 and 23, 1957. In all, more than 2000 observations were made to determine the fluctuations and range of occurrence of contaminant concentrations under a variety of traffic conditions.




**BY MORRIS KATZ, M.SC., PH.D.**
Consultant, Atmospheric Pollution Services, Department of National Health and Welfare, Ottawa, Canada

**R. P. RENNIE, M.SC., CHIEF CHEMIST, AND Z. JEGIER, PH. D.**
Research and Development Division, Canadian National Railways, Montreal, Canada

## THE ST. CLAIR TUNNEL

The St. Clair Tunnel has a length of 6,032 feet and at its mid-point lies 25 feet beneath the bed of the St. Clair River. The tunnel is of tubular shape consisting of a cast iron shell of 20 feet 6 inches diameter, with a cross-sectional area of 312 square feet and a volume of 1,875,000 cubic feet. The slope of the approach and outbound grades in the tunnel is 2 percent. At the time this study was being carried out the tunnel was without forced ventilation.

Due to the features of shape, materials of construction, the cooling effect of the river and of the mass of soil above the tunnel, it was anticipated that there would be little, if any, natural ventilation. Nevertheless, in view of the operational train speed of 15 miles per hour and the fact that the frontal area of a diesel unit is approximately one-half that of the cross-sectional area of the tunnel, a piston action[3] by the train could be expected to draw a certain quantity of diluting air into the tunnel with beneficial effect, thus reducing the extent of air pollution. The tunnel details are shown in Figs. 1 and 2.

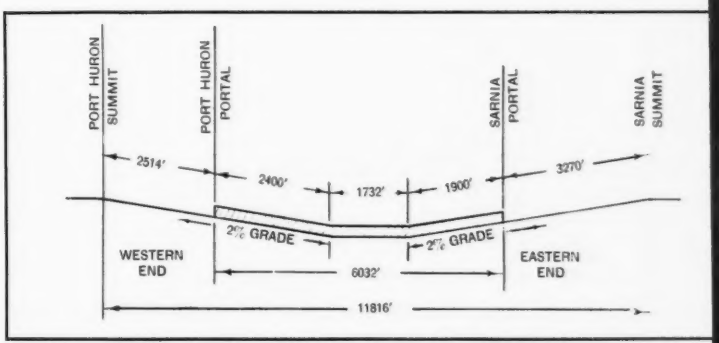



**Fig. 1** *Longitudinal Section of Sarnia — Port Huron Tunnel.*

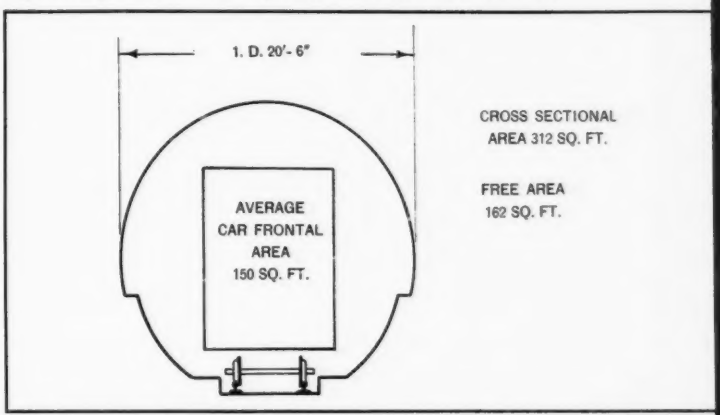



**Fig. 2** *Cross section of tunnel.*

1 Reprinted with permission from the A.M.A. Arch. of Indust. Health, Dec., 1959.

## TABLE I

### Test Stations and their Functions

| *Test Station No.* | *Location* | *Function* |
|---|---|---|
| 1 | Recreation Building — Sarnia Station | Operational Headquarters and Chemical Laboratory |
| 2 | Tunnel Summit Control Tower — Sarnia | Control of train operation in a westerly direction — Sarnia to Port Huron |
| 3 | Pump House — Sarnia Portal | Medical Clinic for taking of blood samples |
| 4 | East Portal — Sarnia | Measurement of air flow in tunnel barometric pressure, temperature |
| 5 | 600 feet inside east portal of tunnel | Sampling of tunnel atmosphere, visibility, temperature and relative humidity |
| 6 | Centre of tunnel | Sampling of tunnel atmosphere, visibility, temperature and relative humidity |
| 7 | 600 feet inside west portal of tunnel | Sampling of tunnel atmosphere, visibility, temperature and relative humidity |
| 8 | West portal — Port Huron | Measurement of air flow in tunnel barometric pressure and temperature |
| 9 | Tunnel Summit Control Tower — Port Huron | Control of train operation in an easterly direction — Port Huron to Sarnia |

## SAMPLING LOCATIONS AND METHODS OF SAMPLING

The lack of forced ventilation and other factors suggested the possibility of wide differences in toxic gas concentrations at different points along the length of the tunnel. In addition, trains enter the tunnel on a downgrade under their own momentum with engines idling and coast to about the midpoint; here power is gradually applied until the locomotive engines are at full power at the outbound grade. In order to contend with this situation it was necessary to collect air samples representing not only average conditions but also fluctuating, peak and residual conditions.

Sample collecting stations were, therefore, chosen at points 600 feet inside the Sarnia and Port Huron portals and at the mid-point of the tunnel to insure that the samples collected would be representative of tunnel conditions. The air sampling stations consisted of wooden, railed-off platforms, erected on the side wall of the tunnel midway between track and roof, each capable of housing a chemist and his assistant with all the necessary air sampling equip-

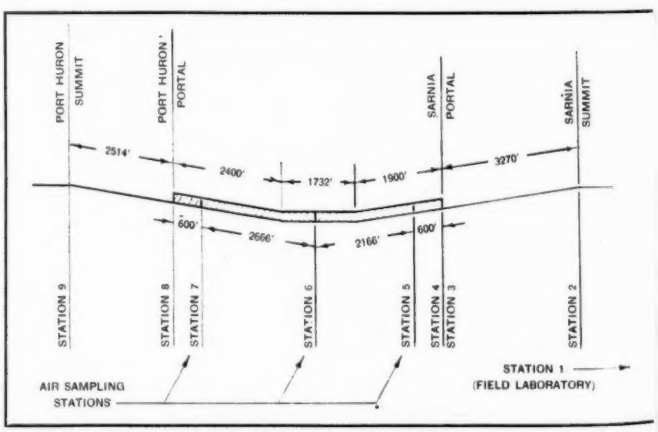



**Fig. 3** *Location of air sampling stations in tunnel.*

ment, including automatic instruments. For a considerable distance on either side of the stations additional electric lighting on the tunnel walls was installed to provide not only the requisite illumination for the operators at the stations, but also to facilitate the observations on visibility conditions in the tunnel.

Each station was fitted with a loud speaker system and telephone for the transmission and reception of messages and instructions to and from the laboratory and the communications section controlling the traffic through the tunnel. A diagram showing the locations of the various test stations is presented in Fig. 3 and the functions of these stations are described in Table I.

During these tests 104 trains passed through the tunnel, from both easterly and westerly directions. Of these 104 trains, 26 trains were powered by locomotives consisting of 4-diesel units, 15 were light engines with 2-diesel units only and, with the exception of idling tests, the remainder were 2-diesel unit trains. Idling tests were conducted with locomotives of 2-diesel units only.

Tests for carbon monoxide were made at the three gas sampling stations during the passage of all 104 trains. Twenty-four trains were designated as "special" trains and during their passage, and at specified periods afterwards, instantaneous samples were collected for the determination of carbon monoxide, nitrogen dioxide, total oxides of nitrogen and formaldehyde. A more limited number of average, ten-minute gas samples were collected by scrubbing through appropriate reagents for the analysis of sulphur dioxide, carbon dioxide, nitrogen dioxide and formaldehyde. During idling tests, snap samples were collected for the determination of carbon monoxide, nitrogen dioxide, total oxides of nitrogen and formaldehyde.

The removal and replacement of sampling equipment were facilitated by a small gasoline-driven rail car. Checks were carried out on the polluting effect of this car with respect to the emission of carbon monoxide during the idling of the car at the midpoint of the tunnel.

"Average" air samples were collected in automatic sequential samplers, each separate intake line having a calibrated orifice for regulating the absorption rate through the absorbents during the collecting period. The sampling mechanism was set in motion at the time the test train locomotive passed the sampling station and was automatically cut out after an absorption period of 10 minutes.

"Instantaneous" air samples were collected by breaking the seals of calibrated one-litre glass tubes that had been evacuated to a pressure of less than 1 mm Hg. The tubes were specially coded for the following determinations: Carbon monoxide, nitrogen dioxide, oxides of nitrogen and aldehyde. The glass inlet seals were snapped off at the following specified times:

1. "Summit" — Train leaving Yard.
2. "Zero time" — Train passing sampling station.
3. "Zero + 3 minutes"
4. "Zero + 6 minutes" —

Samples of blood were obtained from personnel at stations 5, 6, and 7 before and after each 8-hour exposure period by the Medical Staff of the Railway Company and the Occupational Health Division of the Department of National Health and Welfare and analyzed for carboxy-hemoglobin. General medical care was also provided by the medical staff over the 3-day test period.

## METHODS OF ANALYSIS

Several months of investigation, trial, and preparation at the Railway Company's main laboratories were required before a selection of the most suitable methods for analysis could be made. In the choice of methods of analysis, consideration was given to such factors as the conditions to be expected in the tunnel, for example, the possible occurences of comparatively high as well as very low concentrations of toxic gases; the facilities available at the temporary laboratory established at Sarnia; and the effects of storage and transportation on samples that might have to be returned for final analysis to the main laboratories, some 400 miles distant.

After the final choice of methods, it became necessary to thoroughly train a group of chemists and technicians in the specialized sampling and analytical techniques required in the handling of a large number of samples within a reasonable time, with emphasis on both accuracy and speed.

The methods of analysis were as follows:

## CARBON DIOXIDE

Carbon dioxide was absorbed in barium hydroxide containing barium chloride and the excess hydroxide was titrated with oxalic acid using bromothymol blue as indicator[4]. The sensitivity of this method is approximately 20 parts per million by volume.

## CARBON MONOXIDE

(a) Carbon monoxide in snap samples was absorbed in N.B.S. colorimetric indicating gel containing ammonium molydate and palladium

sulphate[5,6]. Standards were prepared from air containing 0.074 percent carbon monoxide as determined by an infra-red spectrometer. This method devised by Shepherd has a sensitivity of approximately 0.2 p.p.m.

(b) Continuous measurements of carbon monoxide concentration were made by the M.S.A. direct indicating instrument which operates on the principle of measurement of heat evolved in the catalytic combustion of carbon monoxide over hopcalite[7]. The scale range of the instrument is from 0 to 0.15% with a sensitivity of approximately 0.002%.

(c) Carbon monoxide in the blood of personnel was determined by means of the micro-gasometric technique of Scholander and Roughton[8]. Some blood samples were shipped by air to Dr. D. V. Bates, Royal Victorial Hospital, Montreal and were analyzed by an infra-red absorption technique[9].

## TOTAL OXIDES OF NITROGEN

Total oxides of nitrogen were determined by the phenol-disulfonic acid method proposed by the Research Department of the Union Oil Company of California[10] as a tentative method to A.S.T.M. Committee D-22 at the time this study was in progress. This procedure has a sensitivity of approximately 5 p.p.m. for total oxides, calculated as $NO_2$.

## NITROGEN DIOXIDE

Nitrogen dioxide was determined by the Saltzman[11] colorimetric method which depends upon the reaction of nitrogen dioxide with sulfanilic acid and N-(1-napthyl)-ethylenediamine dihydrochloride in acetic acid solution. This is an accurate method with a sensitivity of about 0.05 p.p.m.

## FORMALDEHYDE

Formaldehyde was absorbed in an aqueous solution of phenylhydrazine hydrochloride followed by treatment with potassium ferricyanide in alkaline solution[12,13]. The sensitivity of this procedure is approximately 0.2 p.p.m.

## SULPHUR DIOXIDE

Sulphur dioxide was absorbed in a dilute solution containing sulphuric acid and hydrogen peroxide according to the method of M. D. Thomas[14]. Specific conductance was measured by means of an Industrial Instrument Company's Model RC-1 Conductivity Bridge and a conductivity cell with a cell constant of 0.1 reciprocal ohms. As employed here, the method was sensitive to about 0.05 p.p.m.

## OXYGEN

Oxygen was determined by absorption in alkaline pyrogallol using the Orsat apparatus. The procedure was accurate to about 0.25% by volume.

**TABLE II**

**Diesel Fuel Oil — C.N.R. Specification 390-1**

| | |
|---|---|
| Viscosity (S.U.) at 100° F. | 33 Seconds Minimum |
| Flash Point (P.M.) | 125° F. Minimum |
| Cetane No. | 45 Minimum |
| Carbon Residue on 10% Bottoms | 0.15% Maximum |
| Alkali or Mineral Acidity | Nil |
| Sulphur | 0.5% Maximum |
| Water and Sediment | 0.05% Maximum |
| Corrosion Test | Shall Comply |
| Distillation: | |
| 10% distilled at | 460° F. Maximum |
| 50% distilled at | 535° F. Maximum |
| 90% distilled at | 625° F. Maximum |
| Final Boiling Point | 675° F. Maximum |
| Recovery | 98% |

## TABLE III

### Diesel Electric Locomotive — G.M. Road Switcher

| | |
|---|---|
| Engine H.P. | 1750 |
| Number of Cylinders | 16 |
| Stroke Cycle | 2 |
| Bore | 8½″ |
| Stroke | 10″ |
| Compression Ratio | 16:1 |
| Idling Speed | 275 r.p.m. |
| Maximum Speed | 835 r.p.m. |

## SMOKE

Smoke concentrations were determined on a continuous basis by filtration of measured volumes of air through paper tape in A.I.S.I. (Hemeon) smoke samplers[15]. The degree of soiling of the paper was evaluated by optical transmittance of the spots and calculated in COH units per 1000 lineal feet of air filtered. The measurements were made on an hourly cycle.

## PARTICULATE MATTER

Particulate matter was determined by filtration over 8-hour periods through 1106 flash-fired fiberglass webs in high volume samplers[16], followed by gravimetric determination of the fiberglass deposits and expressed as micrograms per cubic meter of air.

## AIR FLOW MEASUREMENTS

Air flow measurements were obtained by the use of calibrated velometers and anemometers. Temperature, relative humidity, and barometric pressure observations were made with all air flow measurements to facilitate the calculation of air flow rates in the tunnel and for calculating concentrations of toxic gases under standard conditions of temperature and pressure.

## TOXIC GAS COMPONENTS OF DIESEL EXHAUST

Diesel exhaust fumes contain a number of toxic gases resulting from the complete or partial combustion of diesel fuel with the excess air used in engine operation[17,18].

The properties of the diesel fuel used in these trials are shown in Table II.

The more harmful components of the exhaust are carbon monoxide, nitrogen dioxide and other oxides of nitrogen, sulphur dioxide, aldehydes, and smoke[19]. The concentrations at which these constituents occur in diesel exhaust largely depend on the fuel/air ratio which in turn varies with the load on the engine[20]. At a given speed, the weight of air intake per unit time is approximately constant, whereas the weight of fuel injected increases as the load on the engine increases. Under normal working conditions the excess air used may be much greater than the stoichiometric requirement.

The trials in the St. Clair Tunnel were conducted using Class GR-17 Road Switchers of General Motors' manufacture. General data for these engines are shown in Table III.

Apart from the results of Berger and McGuire, mentioned earlier, there has been little published information on the exhaust of high horsepower locomotive engines such as those used throughout these trials. The C.N.R. Research Laboratories, however, are conducting investigations in this field, the results of which will be recorded in a future presentation. Preliminary results indicate that the range of toxic gas concentrations in the exhaust gas of newly reconditioned engines of three 1750 HP locomotives under various conditions of load is as shown in Table IV.

## TABLE IV

### Toxic Gas Concentrations in 1750 H.P. Diesel Locomotive Exhaust — P.P.M.

| *Constituent Gas* | *Idling* | *Half Load* | *Full Load* |
|---|---|---|---|
| Carbon Monoxide | 80-110 | 60-105 | 475-480 |
| Nitrogen Dioxide | 65-70 | 250-305 | 330-450 |
| Total Oxides of Nitrogen (Calculated as $NO_2$) | 80-90 | 355-410 | 440-920 |
| Formaldehyde | 3-7 | 2-3 | 4-10 |

## TABLE V

### Mean and Maximum Concentrations of Carbon Monoxide in P.P.M. by Volume For Instantaneous Gas Samples Collected During Passage of Special Test Diesel Trains

| Date 1957 | Time | Station 5 | | | Station 6 | | | Station 7 | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | No. of Samples | Mean | Maximum | No. of Samples | Mean | Maximum | No. of Samples | Mean | Maximum |
| Oct. 18 | 12:01 a.m. — 8:00 a.m. | 8 | 2.7 | 7 | 7 | 2.3 | 5 | 8 | 4.3 | 15 |
| | 8:00 a.m. — 4:00 p.m. | 8 | 6.4 | 20 | 8 | 8.6 | 15 | 8 | 2.8 | 5 |
| | 4:00 p.m. — 12:00 p.m. | 8 | 5.9 | 20 | 8 | 1.3 | 4 | 8 | 1.6 | 5 |
| Oct. 22 | 12:01 a.m. — 8:00 a.m. | 17 | 3.1 | 8 | 24 | 4.0 | 12 | 18 | 2.6 | 12 |
| | 8:00 a.m. — 4:00 p.m. | 18 | 4.4 | 30 | 24 | 6.4 | 20 | 18 | 3.6 | 13 |
| | 4:00 p.m. — 12:00 p.m. | 15 | 2.6 | 7 | 21 | 4.6 | 17 | 12 | 2.6 | 6 (500) |
| Oct. 23 | 12:01 a.m. — 8:00 a.m. | 15 | 1.9 | 5 | 14 | 2.8 | 10 | 16 | 5.3 | 15 |
| | 8:00 a.m. — 4:00 p.m. | 11 | 4.0 | 25 | 11 | 2.9 | 8 | 11 | 5.7 | 20 |
| | 4:00 p.m. — 12:00 p.m. | 9 | 5.4 | 15 | 8 | 4.6 | 17 (150) | 9 | 5.2 | 18 |

*Figures in brackets denote peak values omitted from calculation of mean concentration.

## TABLE VI

### Carbon Monoxide as Percent Saturation of Carboxy-haemoglobin in Blood of Personnel Exposed to Tunnel Atmosphere for 8 hours

| Subject | Exposure Time | HbCO Saturation % | | | Smoking Habit |
|---|---|---|---|---|---|
| | | Before | After | Difference | |
| A | 12:01 a.m. — 8:00 a.m. | 7.50 | 10.60 | +3.10 | Light |
| B | " — " | 3.14 | 2.84 | −0.30 | Moderate |
| C | " — " | 8.50 | 9.35 | +0.85 | Heavy |
| D | " — " | 4.23 | 4.0 | −0.23 | Nil |
| E | " — " | 6.40 | 8.0 | +1.60 | Moderate |
| G | 8:00 a.m. — 4:00 p.m. | 7.10 | 8.0 | +0.90 | Nil |
| H | " — " | 2.25 | 2.4 | +0.15 | Moderate |
| J | " — " | 5.32 | 6.63 | +1.31 | Light |
| K | " — " | 4.17 | 6.85 | +2.68 | Moderate |
| L | " — " | 4.12 | 8.85 | +4.73 | Moderate |
| M | 4:00 p.m. — 12:00 p.m. | 7.15 | 9.30 | +2.15 | Moderate |
| O | " — " | 6.73 | 6.82 | +0.09 | Heavy |
| P | " — " | 2.21 | 2.27 | +0.06 | Nil |
| S | " — " | 2.67 | 9.60 | +6.93 | Light |

## TABLE VII

**Mean and Maximum Concentrations of Nitrogen Dioxide in P.P.M. by Volume For Instantaneous Gas Samples Collected During Passage of Special Test Diesel Trains**

| Date 1957 | Time | Station 5 | | | Station 6 | | | Station 7 | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | No. of Samples | Mean | Maximum | No. of Samples | Mean | Maximum | No. of Samples | Mean | Maximum |
| Oct. 18 | 12:01 a.m. — 8:00 a.m. | 8 | 1.4 | 3.6 | 8 | 0.6 | 1.5 | 8 | 1.6 | 4.2 |
| | 8:00 a.m. — 4:00 p.m. | 8 | 0.9 | 2.3 | 8 | 0.4 | 0.8 | 8 | 1.8 | 8.4 |
| | 4:00 p.m. — 12:00 p.m. | 8 | 0.8 | 2.0 | 6 | 0.4 | 0.6 | 4 | 0.4 | 0.7 |
| Oct. 22 | 12:01 a.m. — 8:00 a.m. | 8 | 1.8 | 9.5 | 14 | 2.0 | 10.0 | 8 | 2.5 | 13.0 |
| | 8:00 a.m. — 4:00 p.m. | 8 | 0.8 | 1.5 | 13 | 2.8 | 12.0 | 8 | 1.2 | 3.0 |
| | 4:00 p.m. — 12:00 p.m. | 8 | 0.5 | 3.5 | 13 | 2.7 | 11.5 | 6 | 2.6 | 15.0 |
| Oct. 23 | 12:01 a.m. — 8:00 a.m. | 10 | 0.6 | 1.8 | 10 | 0.7 | 1.9 | 10 | 2.3 | 9.5 |
| | 8:00 a.m. — 4:00 p.m. | 4 | 1.8 | 5.3 | 3 | 0.7 | 0.9 | 3 | 2.7 | 6.3 |
| | 4:00 p.m. — 12:00 p.m. | 4 | 2.3 | 5.5 | 4 | 0.5 | 0.9 | 4 | 0.6 | 0.8 |

## RESULTS

Most of the analytical data on the extent of contamination of the St. Clair Tunnel atmosphere during the passage of diesel trains in the trial period are presented in summary in Table V — XI. As a guide to assessment of the significance of the results, the threshold limit values for a list of contaminants adopted at the 1958 Annual Meeting of the American Conference of Governmental Industrial Hygienists[21] are of interest. For the gases under consideration here, the following are the respective MAC values in p.p.m. by volume: —

| | |
|---|---|
| Carbon dioxide | 5000 |
| Carbon monoxide | 100 |
| Oxides of nitrogen (Calculated as $NO_2$) | 25 |
| Nitrogen dioxide | 5 |
| Sulphur dioxide | 5 |
| Formaldehyde | 5 |

The value for oxides of nitrogen (excluding nitrous oxide) represents a threshold limit formerly in use that has been omitted from lists of MAC values within recent years. However, it is still useful as a rough guide although no recommendation has been proposed, as yet, for nitric oxide, NO. The current limit for nitric acid is 10 p.p.m. No threshold limits have been established for smoke or nonspecific particulate matter. As regards overall contamination of the air in more or less confined spaces, it is customary to maintain the oxygen concentration at a level greater than 20.5 percent.

Concentrations of carbon monoxide in the tunnel atmosphere were, in general, low, as shown by the data for snap gas samples in Table V. Apart from two exceptional values of 500 and 150 p.p.m., the maximum concentrations were not higher than 30 p.p.m. and the highest mean was only 8.6 p.p.m. A considerable number of observations of carbon monoxide levels were made at each sampling station with the direct indicating instruments which were operated on a continuous basis. Most of these readings were negative owing to the insensitivity of the instruments in this low concentration range. Maximum values were recorded occasionally in the range of 10 to 70 p.p.m., with one reading as high as 170 p.p.m.

Blood samples were obtained from a number of subjects employed at the gas sampling stations, before and after each 8-hour working shift, and analyzed for carboxy-hemoglobin content with results as shown in Table VI. The percentage HbCO saturation levels in subjects prior to exposure to the tunnel atmosphere are roughly indicative of smoking habits, among other factors. After exposure there was some bias towards higher HbCO saturation levels in 50 percent of the 14 subjects listed in Table VI. The remainder showed virtually no change within the limits of $\pm$ 1% HbCO content. These results are in substantial agreement with the fluctuating but generally low average levels of carbon monoxide found in the tunnel environment.

The nitrogen dioxide concentration levels in the tunnel atmosphere from analyses of 204 snap samples

## TABLE VIII

**Mean and Maximum Concentrations of Total Oxides of Nitrogen in P.P.M. by Volume Calculated as $NO_2$, for Instantaneous Gas Samples Collected During Passage of Special Test Diesel Trains**

| *Date 1957* | *Time* | *Station 5* | | | *Station 6* | | | *Station 7* | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | *No. of Samples* | *Mean* | *Maximum* | *No. of Samples* | *Mean* | *Maximum* | *No. of Samples* | *Mean* | *Maximum* |
| Oct. 18 | 12:01 a.m. — 8:00 a.m. | 7 | 25.8 | 56 | 7 | 28.0 | 49 | 7 | 23.3 | 40 |
| | 8:00 a.m. — 4:00 p.m. | 7 | 21.6 | 35 | 7 | 20.6 | 44 | 8 | 25.7 | 46 |
| | 4:00 p.m. — 12:00 p.m. | 8 | 17.7 | 27 | 7 | 18.2 | 26 | 7 | 14.8 | 29 |
| Oct. 22 | 12:01 a.m. — 8:00 a.m. | 7 | 18.5 | 28 | 14 | 19.2 | 34 | 5 | 28.5 | 38 |
| | 8:00 a.m. — 4:00 p.m. | 4 | 19.5 | 28 | 9 | 28.2 | 48 | 3 | 16.7 | 23 |
| | 4:00 p.m. — 12:00 p.m. | 7 | 15.6 | 38 | 14 | 30.8 | 54 | 8 | 28.2 | 64 |
| Oct. 23 | 12:01 a.m. — 8:00 a.m. | 9 | 38.4 | 67 | 7 | 33.7 | 52 | 4 | 35.9 | 55 |
| | 8:00 a.m. — 4:00 p.m. | 4 | 33.1 | 40 | 3 | 19.6 | 25 | 3 | 28.8 | 46 |
| | 4:00 p.m. — 12:00 p.m. | 4 | 26.5 | 38 | 4 | 16.2 | 23 | 3 | 9.1 | 11 |

collected during the passage of 24 gas-test diesel trains varied as shown in Table VII. Mean values were distributed between 0.4 to 2.8 p.p.m. and the extremes of maximum concentration fell within a low of 0.6 and a peak of 15.0 p.p.m. Time-average samples collected over periods of 10 minutes each ranged in concentration from 0.1 to 0.4 p.p.m.

Mean and maximum levels of concentration for total oxides of nitrogen (calculated as $NO_2$) were found to be distributed as shown in Table VIII, from analyses of 177 snap samples collected during the passage of the test trains. The mean values varied from 9.1 to 38.4 p.p.m. and the peak instantaneous concentration reached a level of 67 p.p.m.

The considerable difference in concentration levels between nitrogen dioxide and total oxides of nitrogen is due mainly to the presence of nitric oxide in diesel exhaust gas. As mentioned by Davis and Holtz[19], at the high temperatures of combustion in a diesel engine the reaction $N_2+O_2=2\ NO$ takes place. Depending upon temperature and oxygen concentration, the exhaust gas may contain initially only nitric oxide, or a mixture of nitric oxide and nitrogen dioxide.

Furthermore, a number of chemical reactions occur in mixtures of nitrogen dioxide, nitric oxide, oxygen and water vapour[22,23]. As a consequence nitric oxide is converted to nitrogen dioxide or nitrous acid according to the reactions:

$$2NO + O_2 = 2NO_2$$
$$NO + NO_2 + H_2O = 2HNO_2$$

At equilibrium, in the case where the concentrations of nitric and nitrogen dioxide are equal and water vapour is present to the extent of about 2 percent, the nitrous acid produced would be 19 percent of the concentration of nitric oxide.

The reaction between nitric oxide and oxygen to form nitrogen dioxide follows the rate law,

$$-\frac{d(NO)}{dt} = K\,[NO]^2\,[O_2]$$

Since the concentration of oxygen in the tunnel was found to be essentially constant, the approximate time required for the conversion of one-half of the nitric oxide at ordinary temperatures may be calculated from the equation given by Smith[24], $\frac{1}{x_2} - \frac{1}{x_1} = 0.0144t$, where t is the time in hours, $x_1$ and $x_2$ are the initial and final concentrations of nitric oxide in p.p.m.

In the case of nitric oxide at an initial concentration of 10 p.p.m., the time required for one-half of the nitric oxide to be converted to $NO_2$ is about 7 hours and for an initial concentration of 50 p.p.m., the half life is approximately 1.4 hours.

Since the primary oxidation product of the nitrogen in the air supplied with the fuel to a diesel engine is

## TABLE IX

### Mean and Maximum Concentrations of Formaldehyde in P.P.M. by Volume for Instantaneous Gas Samples Collected During Passage of Special Test Diesel Trains

| Date 1957 | Time | Station 5 | | | Station 6 | | | Station 7 | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | No. of Samples | Mean | Maximum | No. of Samples | Mean | Maximum | No. of Samples | Mean | Maximum |
| Oct. 18 | 12:01 a.m. — 8:00 a.m. | 8 | 3.0 | 3.3 | 8 | 3.3 | 3.9 | 8 | 3.4 | 3.9 |
| | 8:00 a.m. — 4:00 p.m. | 8 | 5.4 | 7.9 | 8 | 3.5 | 4.4 | 8 | 3.5 | 5.1 |
| | 4:00 p.m. — 12:00 p.m. | 8 | 5.7 | 8.3 | 8 | 5.0 | 6.0 | 8 | 3.7 | 5.1 |
| Oct. 22 | 12:01 a.m. — 8:00 a.m. | 8 | 2.3 | 3.7 | 14 | 3.5 | 6.3 | 8 | 2.4 | 3.4 |
| | 8:00 a.m. — 4:00 p.m. | 8 | 5.8 | 8.3 | 14 | 2.9 | 4.3 | 8 | 3.0 | 3.9 |
| | 4:00 p.m. — 12:00 p.m. | 8 | 2.0 | 3.1 | 14 | 2.3 | 3.7 | 8 | 4.9 | 8.9 |
| Oct. 23 | 12:01 a.m. — 8:00 a.m. | 9 | 3.0 | 4.7 | 9 | 2.8 | 3.9 | 10 | 2.8 | 3.3 |
| | 8:00 a.m. — 4:00 p.m. | 4 | 2.0 | 2.6 | 3 | 1.5 | 1.8 | 4 | 2.6 | 3.1 |
| | 4:00 p.m. — 12:00 p.m. | 4 | 2.0 | 2.7 | 4 | 1.2 | 2.0 | 4 | 1.4 | 2.2 |

nitric oxide, the above calculations make it clear that the concentration of nitric oxide in the tunnel atmosphere would tend to be considerably greater than that of nitrogen dioxide. The analytical results on total oxides of nitrogen in relation to nitrogen dioxide in the tunnel atmosphere are, therefore, indicative of the relatively slow rate of oxidation of NO to $NO_2$. At the concentration levels shown in Table VIII, several hours would be required to convert 50 percent of the nitric oxide to the much more toxic nitrogen dioxide. In the meantime, most of the pollution would be swept out of the tunnel by the natural ventilation caused by the piston effect of a passing train and the induced draft that follows.

The extent of contamination of the tunnel atmosphere by aldehydes (calculated as formaldehyde)

## TABLE X

### Concentrations of Particulate Matter in the Tunnel Atmosphere in Micrograms per Cubic Metre of Air from High Volume Filter Samples

| Date, 1957 | Time | Station 5 | Station 6 | Station 7 |
|---|---|---|---|---|
| Oct. 18 | 12:01 a.m. — 8:00 a.m. | 900 | — | 1800 |
| | 8:00 a.m. — 4:00 p.m. | 1100 | 1400 | 1600 |
| | 4:00 p.m. — 12:00 p.m. | 1100 | 1100 | 1100 |
| Oct. 22 | 12:01 a.m. — 8:00 a.m. | 1500 | 1200 | 1700 |
| | 8:00 a.m. — 4:00 p.m. | 2000 | 1600 | 2300 |
| | 4:00 p.m. — 12:00 p.m. | 1700 | 1100 | 1500 |
| Oct. 23 | 12:01 a.m. — 8:00 a.m. | 2200 | 1300 | 1600 |
| | 8:00 a.m. — 4:00 p.m. | 1500 | 1200 | 1000 |
| | 4:00 p.m. — 12:00 p.m. | 2000 | 900 | 1100 |

during the passage of the gas-test trains is shown by the data in Table IX for 213 snap samples. The mean concentrations varied from 1.2 to 5.8 p.p.m. and the highest instantaneous value reached a level of 8.9 p.p.m.

With respect to other gases, the highest concentration of sulphur dioxide was found to be 0.65 p.p.m. and the means of six sets of (10-minute) samples totalling 54 separate determinations ranged from zero to 0.13 p.p.m. The carbon dioxide concentrations in 49 samples over 10-minute sampling periods were generally less than 625 p.p.m. and the maximum level attained was 900 p.p.m. A number of tests for oxygen content of the tunnel atmosphere indicated no deficiency in this respect as the concentration varied between the limits of 20.5 to 20.8 percent.

Relatively high levels of smoke and particulate matter were found inside the tunnel, especially during periods of train activity, as compared with concentrations usually encountered in urban atmospheres. The mean hourly smoke concentrations for twenty-four-hour periods varied from 5.3 to 10.1 COH units per 1000 linear feet of air, with a maximum hourly concentration of 22.4 COH/1000 ft. Parellel observations at a sampling station outside the tunnel yielded hourly values in the range of less than 0.5 to 4.6 COH per 1000 ft. Particulate matter, on the basis of samples collected by high-volume filtration samplers, varied in concentration over eight-hour test periods from 900 to 2300 micrograms per cubic metre of air, as shown in Table X.

A condensed summary of the mean concentrations of air contaminants found in the tunnel atmosphere over 24-hour test periods is shown in Table XI.

## DISCUSSION

The health hazards associated with the concentrations of air pollutants from diesel exhaust liberated in the tunnel during the passage of trains revolve mainly around the levels of total oxides of nitrogen in relation to nitrogen dioxide, formaldehyde, smoke and particulate matter. Close medical supervision was exercised over all men stationed in the tunnel during the tests and participating in the blood sampling. The physical examinations included pulse and blood pressure readings, condition of the eyes, throat and chest, and investigation of reports as to effects of smoke on visibility and possible irritation or lachrimation. Full medical protection was maintained, in readiness, at the Sarnia portal to the tunnel during all hours when the trials were in progress.

Minor eye and throat irritation was reported on three occasions for brief periods of a few minutes during heavier than usual smoke conditions in the tunnel when tests were being conducted with a train stopped at the midpoint of the tunnel and diesel engines idling. However, the subsequent physical examinations failed to reveal any evidence of toxic after-effects, and this was also true for all personnel engaged in the gas-sampling trials on an 8-hour shift basis around the clock.

The generally negative medical findings are substantiated by the results for carboxy-hemoglobin content of participants at the tunnel sampling stations. Such increases in HbCO content as were found in several subjects after exposure were of the same order of magnitude as might be derived from smoking habits. Furthermore, no abnormalities or other after-effects were found in the physical condition of tunnel participants at any time subsequent to these daily 8-hour exposures.

However the following reasons have led to a recommendation for the installation of appropriate forced-draft ventilation for this tunnel prior to full dieselization of train movement to replace electrical operation. Although average levels of carbon monoxide, carbon dioxide, nitrogen dioxide and sulphur dioxide were well below their respective M.A.C. values (see Table XI) some concern was directed at the concentrations found for total oxides of nitrogen, aldehydes, smoke and particulate matter. Such factors as the possible reactions between aerosol and gaseous contaminants, adsorption of toxic gases on carbon particles and other particule matter, and solubility of gaseous components of exhaust in water droplets and other liquid particulates were given due consideration. Finally, the possibility of joint action or the synergistic effect of a mixture of such contaminants was not over-looked.

In the case of nitrogen oxides, the chronic toxicity of nitrogen dioxide would appear to be fairly well established around the current MAC level of 5 p.p.m. for the 8-hour working day as set by the Committee on Threshold Limits of the American Conference of Governmental Industrial Hygienists. On the other hand, there is very little information available on the true toxicity of nitric oxide since all animal tests must involve mixtures of NO with varying amounts of $NO_2$. The consensus is that NO is at least one-fifth as acutely toxic as $NO_2$ and, consequently, can be tolerated in concentrations five fold or greater than $NO_2$. This would tend to set a possible threshold limit for the mixed oxides of about 25 p.p.m., although no such limit has been recognized by the A.C.G.I.H. within recent years. Furthermore, NO as opposed to $NO_2$, apparently does not possess irritating properties within the levels of concentration under consideration here. At sufficiently high concentrations, NO combines with the blood hemoglobin to form methemoglobin. Although

## TABLE XI

### Mean Concentrations of Air Contaminants for Samples Collected During 24-hour Periods

| | | | October 18, 1957 | | | October 22, 1957 | | | October 23, 1957 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| *Contaminant* | *Type of Sample* | *Station No.* | 5 | 6 | 7 | 5 | 6 | 7 | 5 | 6 | 7 |
| Carbon Monoxide | Instantaneous | No. of Samples | 24 | 23 | 24 | 50 | 69 | 48 | 35 | 33 | 36 |
| | | p.p.m. | 5.0 | 4.1 | 2.9 | 3.4 | 5.0 | 2.9 | 3.8 | 3.4 | 5.4 |
| Nitrogen Dioxide | Instantaneous | No. of Samples | 24 | 22 | 20 | 24 | 40 | 22 | 18 | 17 | 17 |
| | | p.p.m. | 1.0 | 0.5 | 1.3 | 1.0 | 2.5 | 2.1 | 1.6 | 0.6 | 1.9 |
| | 10-minute Average | No. of Samples | 3 | 4 | 3 | 5 | 5 | 5 | 12 | 9 | 10 |
| | | p.p.m. | 0.2 | 0.1 | 0.3 | 0.2 | 0.2 | 0.3 | 0.3 | 0.1 | 0.4 |
| Total Oxides of Nitrogen (Calculated as $No_2$) | Instantaneous | No. of Samples | 22 | 21 | 22 | 18 | 37 | 16 | 17 | 14 | 10 |
| | | p.p.m. | 21.7 | 22.3 | 21.3 | 17.8 | 26.1 | 24.5 | 32.7 | 23.2 | 24.6 |
| Formaldehyde | Instantaneous | No. of Samples | 24 | 24 | 24 | 24 | 42 | 24 | 17 | 16 | 18 |
| | | p.p.m. | 4.7 | 3.9 | 3.5 | 3.4 | 2.9 | 3.4 | 2.3 | 1.8 | 2.3 |
| | 10-minute Average | No. of Samples | 1 | 1 | — | 2 | 1 | 1 | 3 | 6 | 6 |
| | | p.p.m. | 3.4 | 2.9 | — | 2.3 | 2.4 | 2.8 | 2.4 | 3.1 | 2.5 |
| Carbon Dioxide | 10-minute Average | No. of Samples | 5 | 5 | 4 | 3 | 2 | 3 | 8 | 8 | 11 |
| | | p.p.m. | 626 | 458 | 469 | 579 | 615 | 630 | 563 | 536 | 575 |
| Sulphur Dioxide | 10-minute Average | No. of Samples | — | — | — | 6 | 6 | 6 | 12 | 12 | 12 |
| | | p.p.m. | — | — | — | 0.00 | 0.01 | 0.13 | 0.07 | 0.10 | 0.13 |
| Smoke | Hourly spots (24 hour mean values) | COH Units per 1000 ft. of air | 5.3 | 7.2 | 7.7 | 6.2 | 10.1 | 8.4 | 9.3 | 7.2 | 8.7 |
| Particulate Matter | Continuous 8-hourly | Micrograms per Cu. M. | 1000 | 1200 | 1500 | 1700 | 1300 | 1800 | 1900 | 1100 | 1200 |

no blood tests were made for methemoglobin in the present study, later work on diesel exhaust contaminants in another tunnel study as yet unpublished showed that with essentially comparable levels of nitrogen oxides there was no detectable formation of methemoglobin in the blood of exposed subjects. As to the possible presence of higher oxides of nitrogen, such as $N_2O_5$ in the tunnel atmosphere, this is considered to be highly unlikely because of the virtual absence of sunlight (or of ozone) in the tunnel environment.

With reference to formaldehyde, it is commonly accepted that at the levels encountered in this study the action of this contaminant is chiefly irritation of all sensitive tissues contacted, such as the eyes and upper respiratory tract. The 5 p.p.m. threshold limit is sufficiently low to avoid any lung injury. In the case of diesel fumes, it is probable that any eye irritation experienced in such exposure is directly attributable to formaldehyde or higher aldehydes.

Smoke and particulate matter represent nonspecific pollutants that are objectionable above certain minimum levels, depending upon effects on visibility and possible irritation of the respiratory tract. However, the presence of a number of polynuclear aromatic hydrocarbons and other compounds in smoke from the combustion of fuels, including diesel smoke, is an added reason for reducing as far as possible the concentration of smoke and particulates in the tunnel atmosphere.

Toxicity experiments by Pattle et al[25] on the direct exhaust from a diesel engine have indicated that there was no mortality and only mild tracheal and lung damage to experimental animals after a 5-hour exposure to a mixture consisting of 560 p.p.m. CO, 23 p.p.m. $NO_2$, 46 p.p.m. oxides of $N_2$ (calculated as $NO_2$), 16 p.p.m. aldehydes (calculated as HCHO) and 74,000 micrograms per cu.m. of particulates. The visibility in this experiment was only six feet. In other experiments, under different engine operating conditions, with $NO_2$ concentrations of 51 and 43 p.p.m. and oxides of $N_2$ of 209 and 174 p.p.m., but with lower concentrations of carbon monoxide and aldehydes quoted above, the fumes were less acrid but much more lethal, a high proportion of the animals dying during or shortly after the 5-hour exposures.

Recommendations as to ventilation of the tunnel atmosphere, therefore, were based on requirements involving the maintenance of atmospheric contaminants well below MAC limits, including an arbitrary value of 25 p.p.m. for total oxides of nitrogen, and corresponding reductions in smoke and particulate matter, bearing in mind the extent to which the movement of trains augments the scale of natural ventilation.

The displacement of part of the tunnel atmosphere by moving trains results in a piston action which pushes a considerable volume of air ahead of the train whilst a corresponding suction effect draws fresh air into the tunnel at the inbound portal. Smoke and exhaust gases in the form of a trailing wake or "slug" are drawn along with the train. The volume of the tunnel has been estimated at 1.875 million cubic feet. The measured average air displacement per train was 1.54 million cubic feet with minimum and maximum values of 1.0 million and 2.6 million cubic feet during the passage of the special test trains.

Theoretical values for residual levels of toxic gases in the tunnel may be computed by means of the formula [26]

$$C = C_0 e^{-Bt} \quad \text{or} \quad \ln (C/C_0) = -Bt$$

where C is the residual gas concentration, $C_0$ is the initial gas concentration, B is the coefficient of ventilation, and t is the time, Bt represents the number of air displacements required to reduce the gas concentration from $C_0$ to C.

In the case of total oxides of nitrogen with an initial, instantaneous concentration of 47.2 p.p.m. at the midpoint of the tunnel and maximum air displacement at 2.60 million cubic feet, estimation of the residual concentration after dilution by air induced by passage of a train through the tunnel, yields a residual level of 11.9 p.p.m. At minimum air displacement of 1.0 million cubic feet and initial concentration of 25.2 p.p.m., this is reduced by calculation to a residual level of 14.8 p.p.m. The theoretical values for residual levels of toxic gases existing in the tunnel between train passages were confirmed by the results of analyses of gas samples collected prior to entrance of trains into the tunnel.

## SUMMARY

The mean concentrations of toxic gases in the St. Clair Tunnel during the 3-day period of these trials, when all train movement was dieselized, were, in general, below the threshold limits of the A.C.G.I.H. There were no abnormal health effects observed on participants during 8-hour exposures to the tunnel atmosphere, with the exception of some minor cases reported of eye and throat irritation on three brief occasions. Nevertheless, in view of the levels of oxides of nitrogen, aldehyde, smoke and particulate matter attained under conditions of maximum contamination, adequate forced ventilation of this tunnel has been recommended to be installed before full dieselization is introduced to replace electrical operation of trains.

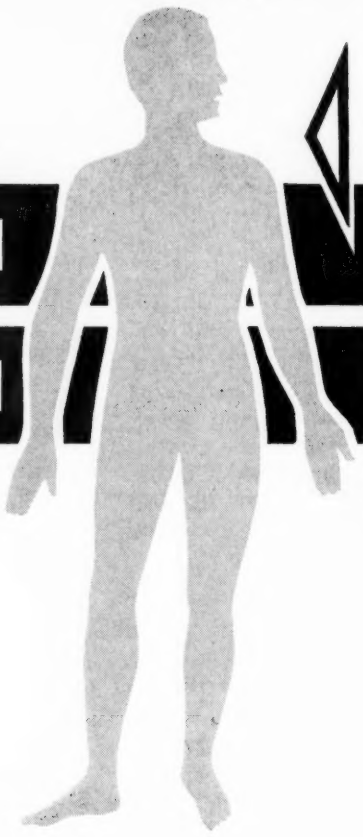
## ACKNOWLEDGEMENT

The assistance provided by the following persons in the investigation is gratefully acknowledged; A. J. de Villiers, M.D., C. R. Ross, G. E. Moore, H. P. Sanderson and J. E. Sinclair of the Occupational Health Division of the Department of National Health and Welfare, Ottawa, D. V. Bates, M.D., Royal Victoria Hospital, Montreal, Dr. H. M. Barrett, Defence Research Board, Ottawa, and various Departments of the Canadian National Railways, including Mr. F. E. King and Staff of the Engineering Section of the Department of Research and Development, Mr. C. I. Biegler and Staff of the Central Region, Operating Department, and J. G. McGuigan, M.D., and Staff of the Central Region, Medical Department.

## REFERENCES

1. Ash, S. H. and L. L. Naus: Use of Diesel Engines in Tunnels. *U.S. Bureau of Mines Information Circular 7222* (October 1942).
2. Berger, L. B. and L. H. McGuire: Observations on the Use of a Diesel Freight Locomotive through a Railway Tunnel, U.S. *Bureau of Mines Report of Investigations*, 3887 (1946).
3. Daugherty, R. L.: Piston Effect of Trains in Tunnels. *A.S.M.E. Transactions*, p. 77 (February 1942).
4. Scott, W. W.: Standard Methods of Chemical Analysis. Norstrand Company, Inc., New York (1945).
5. Shepherd, M.: Rapid Determination of Small Amounts of Carbon Monoxide. *Analytical Chemistry* 19:77 (1947).
6. Shepherd, M., S. Schuhmann, and M. V. Kilday: Determination of Carbon Monoxide in Air Pollution Studies. *Analytical Chemistry* 27:380 (1955).
7. Katz, S. H., D. A. Reynolds, H. W. Frevert, and J. J. Bloomfield: U.S. Bureau of Mines, Technical Paper 355 (1926).
8. Scholander, P. F., and F. J. W. Roughton, *Journal of Biological Chemistry 148:* 551 (1943).
9. Bates, D. V.: The Uptake of Carbon Monoxide in Health and in Emphysema. *Clinical Science* 11: 21-32 (1952).
10. Union Oil Company of California, Research Department: Determination of Oxides of Nitrogen in Gaseous Combustion Products. (March 1957).
11. Saltzman, B. E.: Colorimetric Microdetermination of Nitrogen Dioxide in the Atmosphere. *Analytical Chemistry 26:* 1949 (1954).
12. Kersey, R. W., J. R. Maddock, and T. E. Johnson: Determination of Small Amounts of Formaldehyde in Air. *Analyst 65:* 203 (1940).
13. Tanenbaum, M., and C. E. Bricker: Microdetermination of Free Formaldehyde. *Analytical Chemistry 23:* 354 (1951).
14. Thomas, M. D.: Automatic Apparatus for the Determination of Small Concentrations of Sulphur Dioxide in Air. (*Ind. Eng. Chem., Anal. Ed. 4:* 253 (1932).
15. Hemeon, W. C. L., G. F. Haines Jr., and H. M. Ide: Determination of Haze and Smoke Concentrations by Filter Paper Samplers. *Air Repair* S: 22, 1953.
16. Chambers, L. A.: *American Industrial Hygiene Association Quarterly* 15:290 (1954).
17. Elliot, M. A., R. F. Davis, and R. A. Friedal: Products of Combustion from Diesel Fuel. Proceedings Third World Petroleum Congress, The Hague. Section VII, p. 280-297 (1951).
18. Holtz, John, C., L. B. Berger, M. A. Elliot, and H. H. Schrenk: Diesel Engines Underground, I., Composition of Exhaust Gas from Engines in Proper Mechanical Condition, U.S. *Bureau of Mines Report of Investigations 3508* (1940).
19. Davis, R. F., and J. C. Holtz: Exhaust Gases from Diesel Engines. Chapter 9, p. 74-94, *Problems and Control of Air-Pollution*, F. S. Mallette, Reinhold Pub. Corp., New York (1955).
20. Elliot, M. A.: Review of Bureau of Mines Work on Use of Diesel Engines Underground, U.S. *Bureau of Mines Report of Investigations 4381* (1948).
21. Threshold Limit Values for 1958. *A.M.A. Archives Industrial Health 18* No. 2, p. 178 (August 1958).
22. Cadle, R. D., and H. S. Johnson, Chemical Reactions in Los Angeles Smog. *Proc. Second National Air Pollution Symposium*, Stanford Research Institute, Los Angeles, Calif. (1952).
23. Wayne, L. G. and D. M. Yost: Kinetics of the Rapid Gas-phase Reaction Between NO, $NO_2$ and $H_2O$. *Journal Chem. Phys. 19:* 41 (1951).
24. Smith, J. H. Photocolorimetric Study of the Oxydation of Nitric Oxide at Low Pressures. *Journal Am. Chem. Soc. 65:* 74 (1943).
25. Pattle, R. E., H. Stretch, F. Burgess, K. Sinclair, and J. A. G. Edginton: The Toxicity of Fumes from a Diesel Engine under Four Different Running Conditions. *Brit. J. Industrial Medicine 14:* 47 (January 1957).
26. Morley, M. J., and B. D. Tebbens: The Electrostatic Precipitator-Dilution Method of Flow Measurement. *American Ind. Hyg. Assoc. Quarterly 14:* 4 (December 1953).

# HUMAN HEALTH and them



This conference is concerned with the threats to the health and well-being of man that lurk in his present environment and, specifically, in environmental conditions which, in some important sense, are peculiar to or characteristic of this period in the life of this nation. This is not to say that we have no concern for the larger society of mankind, or that various aspects of these discussions will not impinge upon a much wider segment of human life and experience than that represented in the United States of America. The attempt, however, is to come to grips with certain practical problems that have arisen among us, and to consider the steps which should be taken by those who bear professional or official responsibility for the health and safety of our people now and in the immediate future.

Clearly the health of a nation or a society involves much more than understanding and control of environmental factors of physical and chemical types, and there is some reason at this time for suspecting that the mental and emotional stress of our time may be its most significant feature in relation to the national health. Certainly the national and international climate, with its alarms and excursions, its uncertainties, and its potentialities for imposing sudden and perhaps disastrous social, political and economic consequences upon large segments of our population, is not conducive to ease of mind. Anxiety, apprehension and insecurity are exhibited all around us constantly, not so strikingly, to be sure, as the strident voices of the press and the radio would seem to indicate, but to such an extent, nevertheless, as to be readily apparent. Perhaps the ultimate response to this may be the toughening of our mental and moral fiber. If so, the signs which point to the opposite response are sporadic rather than general, and are to be fleeting rather than abiding.

Be these imponderables as they may, and be our methods of dealing with them as uncertain for some time to come as they now are and have been in the past, there are tasks before us which are within our understanding and competence, tangible threats to human life itself that we can counter, existing information which can and should be applied without further delay, and further knowledge to be gained by methods of investigation that are now available. The situation calls for prompt action for the avoidance of serious and widespread consequences. There is a great need, also, for more sensitive and more effective methods and tools for research, but this is no reason for not employing those we have, thereby developing facts and skills that will open up new

# e modern environment*

**DR. ROBERT A. KEHOE**

Director, Department of Preventive Medicine and Industrial Health, University of Cincinnati, Cincinnati, Ohio.

avenues and methods of approach.

It is not necessary to study vital statistics in order to perceive some of the more glaring hazards of modern urban, or for that matter, rural, life. The severity of these hazards cannot be viewed in their proper proportions without quantitative information, but many departures from an earlier way of life have occurred within one or two generations, and thus the vital statistics of an earlier time, as well as the mechanisms for their collection, are hopelessly inadequate and even archaic. We are aware, however, of a heavy toll of deaths and injuries sustained on the highways and in the homes of the land; of accidental poisonings, especially among children, so numerous as to require regional and local poison centers for the prompt transmission of information and advice; of public apprehension concerning the role of air-borne carcinogenic agents, whether from the partial combustion of petroleum or tobacco, in inducing or contributing to the induction of lung cancer; of heated controversies over the treatment of community water supplies, and the contamination of foodstuffs with insecticides and other foreign substances. And, in a fearsome awe, akin to that imposed by the unknown and unknowable, we contemplate with great uneasiness the dispersion in the air, and the deposition on the land and in the sea, of increasing quantities of radio-active substances. These are the things which are on the horizon of the average citizen.

Looking deeper into the situation with professional eyes, certain other features of the modern environment appear to have graver significance in terms of present and future capacities for harm. Let us sketch two of these in fairly broad strokes.

## OCCUPATIONAL HEALTH IN AN INDUSTRIAL SOCIETY

Through the centuries, man has been called upon frequently to risk his health and even his life, in wresting a living from the land, the sea, the workshop and the mine. But there has never been a time like the present in the history of nations or of men, generally, when man at work has been surrounded by such a multitude of obvious, as well as insidious, dangers to health, limb, organic integrity, and to life itself. Certainly there is a more general and humane concern — a greater fellow feeling — more indeed than ever before, for the health and contentment, as well as the economic status of the laborer. The laborer is also of a very different type than any seen

*Reprinted with permission from Industrial Medicine and Surgery, Volume 28, Number 11, November 1959.

in earlier times. His way of life, in very many respects, differs strikingly from that of previous earners of wages and salaries. Those of our day, especially in our nation, make up the majority of our population. Most of us work for a living, and whether we work with back and arm, or with the observing eye and the trained mind, whether with transport vehicles, earth movers, machine tools, instruments of precision, apparatus for communicating the spoken or written word, or equipment for making calculations at lightning speed, we carry on daily in the midst of a complexity of motion, sound, light effects, equipment, chemicals, and radiant and other forms of energy, that beggars description, and challenges physiological analysis.

Out of this welter of activities come physical and mental wear and tear, physical injuries to sense organs, limbs and integument, and the organic structures of the body as a whole. Death occurs quickly through overt accident, but almost certainly it overtakes many men gradually, reducing their functional efficiency and shortening their lives. Occupational disease kills or disables, at least temporarily, and also takes its toll of life's productive years. By the study of the accidents and illnesses, we learn the cause of injury, disease and death.

But what are the consequences, through the working lifetime, of the frequent, almost daily, impacts of individual and collective insults of minor or subclinical severity? Those with more complacency than curiosity, with more faith in physiological adaptability than can be justified by medical evidence, and with undue comfort derived from crude statistics, will respond to this question with the statement that the people in no previous society have been known to die young in such small numbers, and to live to be old in such large numbers. The easy conclusion from these data is that Americans are healthier now than ever before, and that there can be no very serious threat to health in modern life. But let us look at the facts more thoughtfully and less superficially before we accept this conclusion.

We have reached, if not passed, the zenith of one of the most remarkable and brilliant periods of medical and hygienic history, during which our understanding of infectious diseases was advanced and applied at such a rate that many of the greatest scourges of mankind, in the form of endemic and epidemic diseases, have been almost utterly vanquished. This, the microbiological era of medicine and public health, as it might be called, has wrought a profound change in the practice of medicine, has brought into existence and has cultivated the techniques of preventive medicine, sanitary engineering and public health, and has made revolutionary changes in the statistics of human morbidity and mortality. The diseases in this category have occupied the time and attention of the medical practitioner, investigator and educator, alike, for more than half a century, almost to the exclusion of others. Only in recent years, under the compulsion of the changed frequencies of the causes of illness and death, has attention begun to shift, proportionately, to chronic degenerative diseases and those of the ageing segment of our population.

Meanwhile, the environment of the living, working population of the country has been changing, and the appreciably subdued microbiological threats to human life and health have been replaced by the inanimate threat of the machines and materials which inventive man has made, and by the forms of energy with which he has surrounded himself. The physical basis of modern industry is something new under the sun — and environment made by man which, in the making, has taken so much of his total time, effort, thought and ingenuity as to leave very little for expenditure on learning what might be its ultimate effects upon himself.

As might be expected, the most strategic site for the examination of the industrial environment and of its impact upon man, is within industry itself. Until recently, however, the medical and sanitary sciences have been represented but scantily among the personnel and facilities of industry, and the great proportion of American industrial organizations, especially the preponderant, small units, have operated almost entirely without benefit of preventive medicine and industrial hygiene. Moreover, medical and hygienic research has explored only the surface of the industrial environment; educators in medicine and sanitation have failed, for the most part, to recognize, much less to keep pace with, its existing hygienic problems. While we wait to muster our forces, and to deploy them in greater numbers and in a more effective manner, these problems grow in number and variety.

Only a minute proportion of our industrial organizations have valid medical information concerning the hygienic status of their employees, while the composite picture of the state of health of our general industrial population does not exist even in rough outline. Not only is the incidence of occupational disease unknown, because of the lack of suitable mechanisms for reporting and assembling information that comes to light, but great areas of industry are not under such observation as would provide such information. It is by no means certain, therefore, that the harmful effects of the composite hazards of industry have not exerted important and widespread effects upon the national health, which, because of

the brilliant results of our efforts in the field of microbiological prophylaxis and therapy, may have been masked in our gross statistics of morbidity and mortality.

These comments are not made to incite apprehension, or to point the finger of accusation at any commercial, political or professional group, for indeed it is not likely that the national health has suffered disastrously or irreversibly up to the present time, and if it has been affected significantly, we are all at fault for our lack of vision and our preoccupation. Their point, however, is that we do not know, and we have no present means of knowing, just what has resulted from the profound changes wrought in the industrial environment by modern technology. As these changes progress in their complexity, how are we to know where they are taking us, hygienically speaking, unless we examine them closely, develop methods for their qualitative and quantitative appraisal, and equip ourselves to understand and control their effects? We are, indeed, becoming concerned about ionizing radiation, which has been far better studied and controlled, thus far, than almost any other newly exploited technologic development. Why are we not more deeply concerned about what has been happening to us, largely unobserved and with little anticipatory investigation, during the last half century, within which the environment of the day's work has been revolutionized?

We have been devoting large expenditures, magnificent facilities and the best brains of the country, in increasing numbers and proportions, to the development of new technologic departures from our previous handiwork. Why can we not devote an adequate proportion of the financial and intellectual resources of the country to the guidance of modern industrial technology in the ways of human safety and health? The time has come when we must carry out physiologic and hygienic research hand in hand with technologic research, and when we must develop the methods and techniques of industrial hygiene stepwise with the technology of production. True, this is being done, but only in token. Neither industrial management nor its hygienic advisors can be credited, generally, with a sound grasp of the situation or with plans for handling it.

## PUBLIC HEALTH IN AN INDUSTRIAL SOCIETY

If we are concerned about our industrial population, we have cause also for concern about our population generally, for the industrial environment has become, to a remarkable extent, the national environment. We have called attention to this elsewhere, in commenting on the extent to which the problems of modern medical practice have their origin in industry. The "streets, highways, railways and airways (of the country) are hazardous because of the machinery and speed of transportation, and the great variety of the products transported; its homes and farms are dangerous because of many of the same factors that create industrial hazards — the number and variety of machines, the sources of power, the fuels and refrigerants, the drugs and chemicals that serve agriculture and the modern household; its streams, which are often its general water supplies, are polluted with common sewage, but also with all of the non-volatile substances that comprise the raw, intermediate and finished (chemical) products of industry; its food contains a host of natural substances of which the earth is composed, and variable quantities, as well, of scores of additives, contaminants and chemical substances derived from the modern use of fertilizers and insecticides, or from

the multiplicity of materials employed in processing and packaging our foods; the air of its towns and cities, and often that of its open country side, is burdened with the smoke of dwellings and other buildings, of transportation equipment and of factories, and also with some portion of almost all of modern materials and chemicals, as these are released from the vents and stacks of industry. This new and dangerous environment, which man has created for himself, now provides the challenge to both curative and preventive medicine — a challenge that requires additional types of medical knowledge, new medical skills, and new settings for the application of such knowledge and skill." Let us examine more closely but one feature of this situation, with the idea of indicating some of the methods of investigation that will find application within the general field.

Some hundreds of new chemicals find new uses in American industry annually. These may be new plastics, plasticizers, or additives to alloys, fuels or foods; they may be pesticides, preservatives, solvents, detergents, adhesives or abrasives; or they may be part of the composition of fabrics used for clothing, household draperies and kitchen equipment. Many of these materials come into intimate contact with the people of the nation in their places of work, in their conveyances to and from work, and in their homes. Many of them enter directly into the food of the land, and many others find their way indirectly into the food, the water supply and the atmosphere. In these and other ways these chemicals find their way into the midst of the already complicated chemical reaction mass of the human body.

In speaking of the chemistry of life, one is aware of certain complexities which have been revealed only partially and superficially by biochemical and physiological research. Man is composed in some part of the elements and substances that enter into the composition of the earth. The contributions made by certain mineral elements alone to the mechanisms of life have been investigated at length, but our general knowledge is but fragmentary and often fortuitous. Nevertheless, we endure our ignorance with a certain philosophical equanimity in recognition of the fact that the composition of the earth and the composition of man exist in an intimate historical and biological relationship which is inevitable and not of our making. But what can one think of the artificial situation, in which many new materials, possessed of many, almost an infinity of, possibilities for metabolic modification within the body of man, find their way into it, for better or worse. We shall never be able to anticipate and visualize the harmful potentialities of new materials until we achieve a comprehensive understanding of the role of the normal elements and other components of the living organism. Only on such a comprehensive background can we discover, fairly promptly, the metabolic deviations, if any, induced by new chemicals.

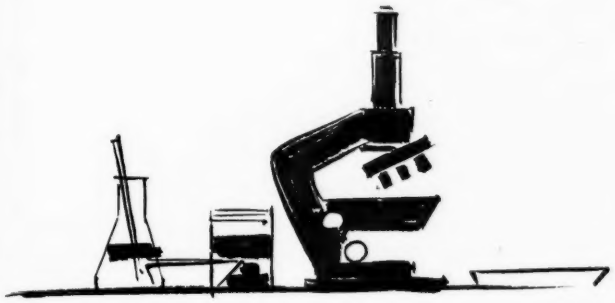
This brings us to consider the foregoing problem from a somewhat different viewpoint. No principle of biology is clearer than that which relates a physiologic response to a quantity of stimulus within appropriate limits of time. The living organism is resistant in varying degrees to varying stimuli, and the stimulus must be adequate to induce a response. It is this principle on which we base the differentiation of harmful versus harmless stimuli in terms of dosage and time. Toxicity, in these terms, does not relate to a specific substance but to a concentration of that substance arrived at within certain limits of time in the effective tissue or chemical system within an organism. A toxic environment, by the same token, is one which, again within certain limits of time, introduces into the organism a harmful quantity of a substance. The means by which this is done may be single or multiple — that is the medium may be the atmosphere, water or food, or any combination of these. The multiple environmental sources of human exposure to chemicals provide a good illustration of the complexities of the problem of public safety. Let us look at such a situation as it relates to a mineral constituent of the earth.

A mineral taken from the earth and brought into commercial use, may pose certain problems of industrial health, from the aspect of the toxicity of one or more of its ingredients. Measures of control can be provided on the basis of certain toxicologic facts, and certain products can be made available in the desired form or forms of commerce, without undue industrial hazard. In the process, constituents of the mineral have been discharged into the air around the production factory. These settle out on the land, thereby increasing its content of the mineral in question. Vegetation which grows out of such soil may take up the mineral from the soil, and the mineral may also be deposited upon the vegetation, which then, if eaten by animals or men, carries more than its natural content of such minerals into their bodies. Such domestic animals and animal products, used as human food, contribute to the total intake of the mineral by man.

At the same time, water-borne waste from the factory is conveyed away in pipes or ditches. This pollutes a stream nearby and affects an entire drainage area to an extent which may be only theoretical on the one hand, or highly significant on the other.

This process, in one form or another, may be multiplied in many or all parts of a country, so that a mineral constituent brought forth out of the earth

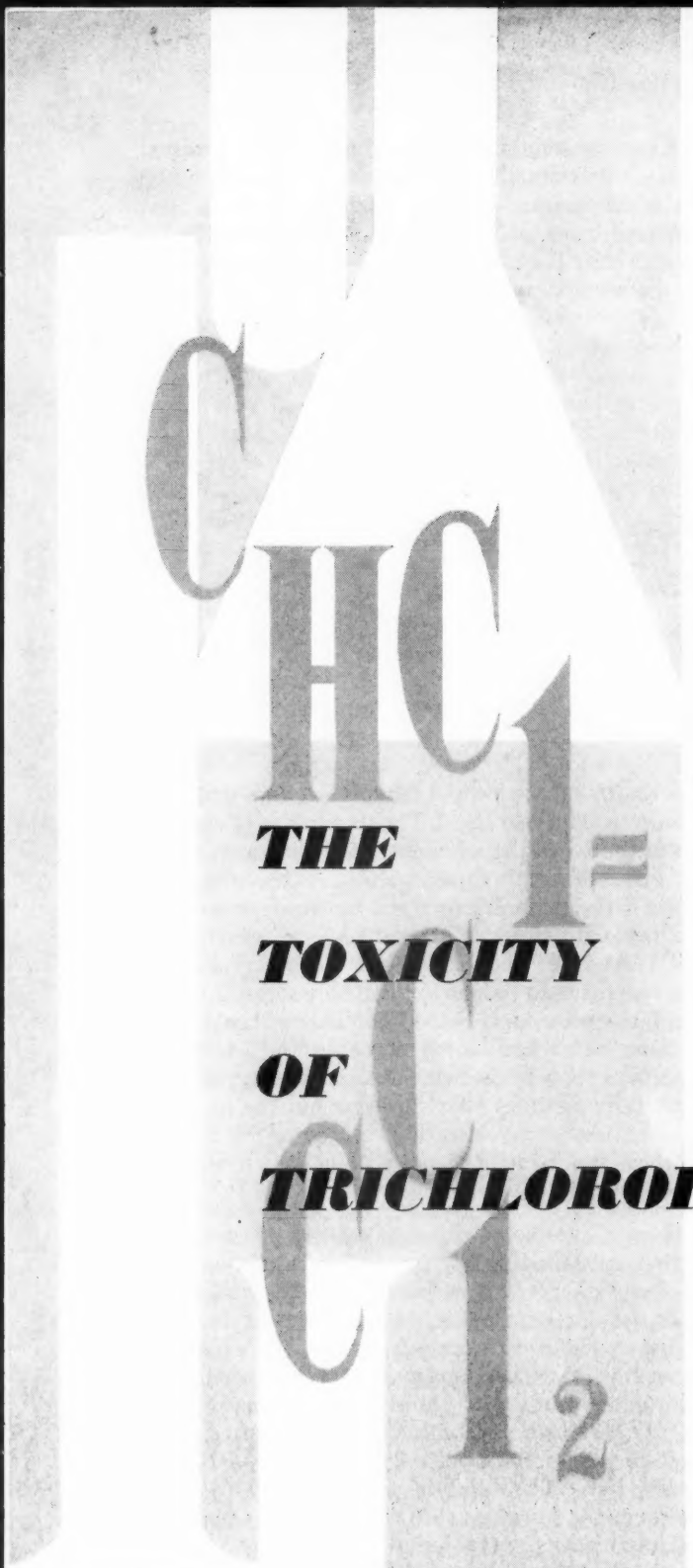
comes to be spread far and wide in the air, in the water and in the food materials of animals and men over a continent, or even over the world. This same process, multiplied still further by the industry, agriculture and the other activities of a country, may so change the balance of nature, with respect to the numbers of the normal constituents of the earth and their proportional representation in the soil, water and atmosphere of the country, as to alter the mineral metabolism of mankind in the area concerned.



The illustration which we have chosen is only one of many that could be taken to follow the course of technologic man in his efforts to make use of the materials of the earth for his own purposes, to wrest from nature secrets that will feed, clothe and house her growing population, that will transport him with ever increasing speed to the four corners of the earth and beyond, and release energy from natural sources and direct it to carry out his bidding. In doing these many things, as has been pointed out elsewhere in great detail, man has changed and is changing further the face of the earth, has altered and is further disturbing the conditions under which life on this planet came into existence and has persisted, with changes and differentiation for some millions of years. It seems likely that man's activities in strictly modern times, as represented in his technologic applications of the natural sciences, have brought about greater environmental changes of more profound physiological significance than have all the generations of men heretofore. During the greater part of historical times man has been contending with natural forces and natural threats to his survival. He has now moved into a period in which his knowledge and power have enabled him to modify the very conditions of his life. The time has come, however, when a much greater and more comprehensive knowledge of the consequences of our changed and changing environment must be had for our safety — perhaps for our very survival.

The efforts made to obtain the necessities of life and then to seek comfort, wealth and power have occupied most of the men of our time and every other time. The men of all ages, who have sought by the methods of their time to cultivate and protect the health of the people, have been few and their resources relatively small. The technology of our time has created a wealth of materials and made available the forces of nature for man's use. At the same time it has failed to bring to these materials and forces the understanding of their biological effects that will keep them under adequate control. The effort must be made to reduce the gap between technology and biology before it is too late. This will take time, studious effort and money: time for the training of experts in the various fields of medicine, hygiene and sanitation; studious effort to ferret out the problems most in need of investigation; money to initiate and catalyze the investigations without which we shall remain in relative ignorance.

It must become an axiom of modern chemical and technologic research that the materials produced and the forms of energy harnessed must be as well understood for their biological potentialities as for their physical and mechanical properties. It is equally important that other man-made factors in the human environment, including those which we cannot foresee at this time, should be examined critically for their harmful potentialities, in the full recognition of certain facts of life and death, which have recently come upon some of us with a shock. It is clear that reckless man can turn loose and build up physical forces which may destroy himself and his kind. It seems probable, however, that the same ingenuity which thus far has enabled him to penetrate other secrets of nature will serve his need when expended in learning more of his own vulnerability and in developing the means for self-protection.



# THE TOXICITY OF TRICHLOROETHYLENE*

BY JONATHAN W. WILLIAMS, PH.D.
WILMINGTON, DELAWARE

Trichloroethylene is used principally as a solvent in degreasing and dry-cleaning processes. It also has many other uses, most of which are based on its excellent solvent action for fats and oils; for example, it finds some application in the paint industry and in the extraction of fats and oils from food products. A few of its applications make use of the pharmacological effects of trichloroethylene; it has been widely used as an anesthetic and to a limited extent as a disinfectant.

Trichloroethylene, $CHCl=CCl_2$, is a volatile liquid, boiling at 87° C., solidifying at −88° C. and having a specific gravity of 1.46 at 20° C. It is completely miscible with alcohol, acetone, ether, and most organic solvents, but is soluble in water only to a very limited degree (0.04% at 25° C.) It is nonflammable and nonexplosive at ordinary room temperature but moderately flammable at much higher temperatures. (The ignition temperature is 410° C.)

Trichloroethylene has an odor resembling that of chloroform. The odor threshold has been stated by Stüber[1] to be 160 ppm, although there is reason to think that for most persons the threshold may be closer to 50 ppm[2].

Trichloroethylene is manufactured by heating 1,1, 2,2-tetrachloroethane with calcium hydroxide. A byproduct which may be formed in this reaction is dichloroacetylene. Unless the preparation is very carefully controlled, the final product may contain appreciable amounts of unchanged tetrachloroethane or dichloroacetylene, or both. Each of these possible contaminants is more toxic than trichloroethylene. In assessing the many conflicting statements in the literature on the toxicity of trichloroethylene, it should be borne in mind that not all preparations studied were equivalent in degree of purity. Exposure to oxygen or strong light will result in gradual decomposition unless a stabilizer such as triethylamine (an acid acceptor) is present. The very irritating dichloroacetyl chloride may be formed by oxidation, and further decomposition will result in the formation of phosgene, hydrogen chloride, and carbon monoxide.

General summaries of the toxicity of trichloroethylene have been published by Browning,[3] Fairhall,[4] von Oettingen,[5] and the Manufacturing Chemists Association.[2]

Dr. Williams is Assistant Director, Haskell Laboratory for Toxicology and Industrial Medicine, E. I. du Pont de Nemours & Company.

*Reprinted with permission from the Journal of Occupational Medicine, Volume 1, Number 10, October 1959.

## TOXICITY — ANIMAL STUDIES

In tests comparing the intravenous toxicity to dogs of trichloroethylene and chloroform, Barsoum and Saad[6] found chloroform to be the more toxic. The minimum lethal doses were 150 mg/kg for trichloroethylene and 90 mg/kg for chloroform.

In inhalation studies with small animals Lazarew[7] found that the acute lethal concentration for the mouse was 7.400 ppm in a two-hour exposure. In a similar test Carrieu[8] found the lethal concentration for guinea pigs to be 27,000 ppm. The narcotic concentration was of the order of 3,700 to 4,600 ppm.[7,9,10,11] Taylor[11] found that 0.2% produced slight drowsiness and from 0.4 to 0.5% produced deep narcosis in rats. McCord[12] found that the narcosis threshold for rabbits was 500 ppm. Taylor[11] found that trichloroethylene produced narcosis in animals with no profound effect on the respiratory or vascular system. The interval between the narcotic dose and the lethal dose was shown to be relatively wide; Genevois[13] stated that the lethal dose was about two and one-half times the narcotic dose.

Acute poisoning by trichloroethylene was shown by Barrett, Maclean, and Cunningham[14] to have little or no specific effect on the liver or kidneys of cats or rats; the usual pathological finding was acute congestion of all internal organs. With guinea pigs there were slight degenerative changes in the livers of some animals.[14] Herzberg[15] and Meyer[16] found fatty infiltration of the liver in dogs subjected to trichloroethylene vapors; Barsoum and Saad[6] found similar changes in the livers of dogs receiving trichloroethylene intravenously.

In experiments with rats by Adams, Spencer, Rowe, McCollister, and Irish,[17] determinations were made of the severest single exposures permitting survival of 100% of the animals. It was found that the rats survived exposure to 20,000 ppm for a period of 0.3 hour, 12,000 ppm for 0.6 hour, 9,600 ppm for 0.8 hour, 6,400 ppm for 1.4 hours, 4,800 for 1.4 hours, and 3,000 ppm for 8 hours. Similar experiments with monkeys showed survival of all animals exposed to 400 ppm for seven hours, with rabbits 200 ppm for seven hours, and with guinea pigs 100 ppm for seven hours. It was reported that the only toxic effect of importance during the single acute exposures was a depressant action on the central nervous system, or anesthetic action.

In a study of chronic inhalation exposure using cats as the experimental animals, Lehmann[10] found that an exposure to 760 ppm for six hours a day over a period of 10 to 17 days produced drowsiness and loss of weight and a few deaths. Lehmann and Schmidt-Kehl[18,19] showed that cats exposed to 3,500 ppm suffered loss of equilibrium after 75 minutes, became prostrate within 235 minutes, exhibited slight narcosis after about 275 minutes, and were in a state of deep narcosis within 300 minutes after the beginning of the exposure. When the concentration to which the cats were exposed was 22,500 ppm, loss of equilibrium was immediate, prostration occurred after four minutes, slight narcosis after seven minutes, and deep narcosis after about eight minutes. Barrett, Maclean, and Cunningham[14] found that the exposure of guinea pigs and rabbits to 1,200 ppm of trichloroethylene for 473 hours was without significant effect.

Seifter[20] exposed dogs to 500 to 750 ppm for four to eight hours daily, five to six days per week. The clinical signs evidenced by the dogs included lethargy, anorexia, nausea, vomiting, and loss of weight. There was a gradual decrease of up to 10% in the total number of erythrocytes in these dogs. The dogs showed liver dysfunction within three weeks as indicated by the sulfobromophthalein sodium (Bromsulfalein) test. The urine was found to contain free chloral. The liver showed depletion of glycogen and hydropic parenchymatous degeneration. However, microscopic study of liver sections made from dogs that were allowed to recuperate until the bromsulfalein test was normal showed no signs of liver injury.

Adams, Spencer, Rowe, McCollister, and Irish[17] exposed four different animal species seven hours daily, five days a week, to trichloroethylene vapor for about six months. The maximum concentrations of trichloroethylene found to be without adverse effect on the animals were as follows: the monkey, 400 ppm; the rat and the rabbit, 200 ppm; the guinea pig, 100 ppm.

It has been demonstrated that trichloroethylene may exert a toxic effect by absorption through the skin. In experiments by McCord[12] with rabbits, it was found that the application of 3.8 ml/kg of body weight three times daily produced some deaths after five days. A similar program with doses of 3.6 ml/kg failed to produce fatalities.

It has been stated by Mogilevskaya[21] that in addition to the narcotic effect, trichloroethylene may specifically attack the sensory fibers and the visual nerve, leading to blindness. He stated that it could cause severe hepatitis and damage to the respiratory organs, possibly due to the formation of hydrochloric acid and phosgene. The wide discrepancy between these statements and those of other workers causes one to wonder about the purity of the sample studied by Mogilevskaya.

## CLINICAL EXPERIENCE WITH MAN

The chief concern with the use of trichloroethylene in industry is the danger of narcosis which may

follow prolonged exposure to high concentrations. Most of the fatal cases recorded in the literature have involved unconsciousness as an early manifestation. Stüber[1] in 1931 recorded 284 cases of presumed exposure to trichloroethylene, 25 of which were fatal, as having occurred in German industry. Browning[3] stated that between 1938 and 1948 there were 224 cases of "gassing" from trichloroethylene reported to the Chief Inspector of Factories in Great Britain. Of these 224 cases, 10 were fatal. No comprehensive summary of American industrial experience has been published, although Quadland[22] has surveyed the medical literature from 1932 through 1943, and found 11 cases described, two of which had fatal consequences. Striker, Goldblatt, Warn, and Jackson[23] found in an investigation of 304 cases of the use of trichloroethylene as an anesthetic that there were no lasting harmful consequences.

The symptoms usually exhibited in the acute cases were signs of inebriation, giddiness, headache and loss of appetite. Stüber[1] found 10 cases of isolated trigeminal paralysis in the 284 cases studied. Disturbance of cardiac action has been reported. There were also nine cases showing optical disturbances due to lesions of the optic nerve, including two cases of blindness from optic atrophy. Gastric disorders were fairly frequent complaints. These included nausea, vomiting, and loss of appetite. Cases have been described where abdominal cramps and diarrhea were persistent.

In a review of five fatal cases of trichloroethylene exposure, Kleinfeld and Tabershaw[24] stated that the mechanism of death in four of the five cases was most probably ventricular fibrillation. These four workmen died suddenly, either at work or shortly after leaving the plant. Autopsies showed no gross anatomical change other than congestion of the viscera, but analysis of tissues revealed the presence of appreciable amounts of trichloroethylene. There have been other reports of cardiac arrythmia following exposure to high concentrations of trichloroethylene.[25-28] These findings are in accord with the principle[29] that all fat solvents, including both hydrocarbons and chlorinated hydrocarbons, tend to sensitize the myocardium to the action of epinephrine (adrenalin), with resulting ventricular fibrillation.

It has been generally agreed that the liver is not a prime target organ. Stüber[1] went so far as to state that the liver is never affected. However, several other workers have reported that hepatitis and cirrhosis of the liver could be caused by trichloroethylene but, it appeared, much less frequently than by tetrachloroethane, carbon tetrachloride, or chloroform.

Other effects noted have been skin lesions and mucous membrane lesions. The skin effects were reddening and burning which could occur after comparatively short contact. However, serious inflammation with blistering occurred only when free evaporation was prevented by covering the skin. Instances have been reported by Stüber[1] and McCord[12] of irritation of the air passages. It was considered that this was possibly due, not to trichloroethylene itself, but to a product of its decomposition, such as hydrochloric acid.

## METABOLISM OF TRICHLOROETHYLENE IN ANIMALS AND MAN

Animal experiments carried out by Barrett and Johnston[30] and Barrett, Cunningham, and Johnston[31] showed trichloroacetic acid to be an end-product of trichloroethylene metabolism. Estimation of the percentage of trichloroethylene inhaled which was excreted as trichloroacetic acid in the urine of dogs showed it to be from 5% to 8%. A single experiment on a human being indicated that trichloroethylene was similarly metabolized in the human organism. Powell[22] suggested that routine blood and urine tests for trichloroacetic acid could be used as measures of harmful exposures to trichloroethylene in industry. Ahlmark and Forssman[33] showed that the extent of exposure to trichloroethylene may be evaluated by determining the trichloroacetic acid content of the urine. In an examination of 80 workers exposed to trichloroethylene vapors in the range 23-59 ppm Grandjean et al.[34] found the average ratio of urinary trichloroacetic acid expressed in milligrams per liter to atmospheric trichloroethylene expressed in parts per million to be 3 to 1. Animal experiments carried out by Fabre and Truhaut[35] indicated that both trichloroethylene and the trichloroacetic acid derived from it might be deposited in the endocrine glands, the kidneys, the brain, the blood, and particularly in the spleen and lungs.

Further studies of metabolism of trichloroethylene in the animal organism have been carried out by Forssman, Owe-Larsson, and Skog.[36] They found that, with dogs and rats, subcutaneous injection of trichloroethylene, tri-chloroethanol, or chloral resulted in the excretion of only a small percentage as trichloroacetic acid. On the other hand, injection of the sodium salt of trichloroacetic acid was followed by excretion of from one-third to one-half of the acid injected. Another group of animals was given a suspension of 25 mg. of tetraethylthiuram disulfide (antabuse) in olive oil by stomach tube. This material was known to be an inhibitor of oxidation of aldehydes, and thereby a potentiator of the toxicity of aldehydes and the corresponding alcohols. The animals receiving the tetraethylthiuram disulfide showed a

marked decrease in the excretion of trichloroacetic acid after injection of trichloroethylene or trichloroethanol, a smaller decrease after the injection of chloral, and no change after the administration by mouth of sodium trichloroacetate. The results of this study indicated that the route of metabolism of trichloroethylene in the animal organism is that of an oxidation via trichloroethanol and chloral to trichloroacetic acid.

## TOXICITY OF SOYBEAN MEAL AFTER EXTRACTION WITH TRICHLOROETHYLENE

An unusual sideline to the toxicity of trichloroethylene developed in connection with its use as an extractant for the oil from soybean meal. The trichloroethylene-extracted soybean meal has been shown to cause a refractory hemorrhagic aplastic anemia when fed to cattle. On the other hand, soybean meal which has been extracted with hexane or benzene or even with carbon tetrachloride has never shown this type of toxicity.

The first reported poisoning of cattle with trichloroethylene-extracted soybean meal occurred in southern Scotland in 1912 and was reported by Stockman[37] in 1916. Other widespread outbreaks[38] occurred in Germany and Holland in 1923-1925 and in the United States, Italy, and Japan during the period 1947 to 1952.

Similar poisoning of horses[39] and a related poisoning of sheep[40] have been reported, but the toxicity does not appear to extend to other species.

The characteristic blood picture in trichloroethylene-extracted soybean meal has been found to be a leukopenia accompanied by a relative lymphocytosis with suggested damage to the bone marrow.[38]

Various theories have been advanced to account for this toxic action. These include, among others, the destruction of vitamins,[41] a phosphatide deficiency, and a type of urease poisoning.[38] It has been demonstrated that trichloroethylene itself does not produce this particular toxic response.

It has recently been shown by Picken and coworkers[42] that a highly toxic factor is formed by the reaction of trichloroethylene with the protein fraction of the meal. This factor has been concentrated, and its potential for causing hemorrhagic aplastic anemia has been demonstrated. A product, S-(dichlorovinyl)-L-cysteine, synthesized by these workers from trichloroethylene and cysteine, showed the typical toxicity syndrome in tests with calves, and showed physical properties in paper chromatography tests similar to those demonstrated by the toxic protein fraction from tri-chloroethylene-extracted soybean meal. Picken suggested that S-(dichlorovinyl)-L-cysteine may be related in structure to a part of, or may possibly be, the toxic principle in tri-chloroethylene-extracted soybean meal.

## MAXIMAL ACCEPTABLE CONCENTRATION

The primary consideration in the prevention of toxic effects due to trichloroethylene exposure is the control of the vapor concentration in the working atmosphere. The American Conference of Governmental Industial Hygienists[43] and the Hygienic Guides Committee of the American Industrial Hygiene Association[44] have adopted 200 ppm as a threshold limit for eight hours' exposure daily five days a week. The adequacy of this Maximal Acceptable Concentration (MAC) has been questioned by some workers,[45,46] and there is lack of agreement on the interpretation of the MAC value as a ceiling or as an average concentration.

Maximal Acceptable Concentrations have been set elsewhere over a wide range. For example, the firm of Imperial Chemical Industries, Ltd., in Egland has adopted 200 ppm;[47] the Russian figure is 9 ppm, and in Italy the value commonly used is 100 ppm.[46] Arguing on the basis of trichloroacetic acid excretion figures, Ahlmark and Friberg[48] in Sweden have stated the average daily exposure to trichloroethylene should not exceed 30 ppm.

In reviewing the European work on trichloroethylene, Stokinger[46] has raised questions concerning the purity of the trichloroethylene and the analytical methods used abroad to determine trichloroethylene concentrations. He also mentioned the lack of evidence in the United States of injury among workers exposed chronically to trichloroethylene for a period of many years.

## SAFETY AND MEDICAL CONSIDERATIONS

Workers in trichloroethylene areas should be provided with eye protection of the safety goggle type. In some situations complete face protection may be desirable and can be provided by means of plastic face shields.

Inasmuch as sustained or repeated skin contact with liquid trichloroethylene may produce dermatitis, it is important that any trichloroethylene on the skin should be immediately removed by washing with soap and water. Likewise, contaminated clothing should be removed promptly and laundered or thoroughly dried before reuse. In areas where the worker is vulnerable to trichloroethylene splashes, impervious clothing, such as that made of Neoprene, may be worn.

Respiratory protection is of the highest importance in instances where it is necessary for workers to clean

out tanks which have contained trichloroethylene and, likewise, in decontaminating areas following spills. Several forms of respiratory protection may be considered. The self-contained breathing apparatus involving a supply of oxygen is useful where it is necessary for the workers to move about freely. The use of an oxygen-generating chemical, such as $K_2O_4$, in the self-contained breathing apparatus is not recommended, since a reaction forming dichloroacetylene may occur. The airline mask is suitable for use where conditions will permit safe escape in case of failure of the compressed air supply. An industrial canister type gas mask fitted with the proper canister for absorbing trichloroethylene vapor will afford short-term protection against concentrations of trichloroethylene not exceeding 2% by volume. It must not be used when the oxygen content of the air is less than 16% by volume, and the use must always be for a relatively short exposure period only.[2]

A person inhaling an excessive amount of trichloroethylene vapor within a short period of time will show symptoms which are associated with most volatile anesthetics. At first there may be irritation of the eyes, nose, and throat, followed by dizziness, nausea, vomiting, and gradual loss of consciousness. If one is exposed to a high concentration over a relatively long period of time, there may be a complete suppression of the pain sense and loss of muscular activity.

Chronic exposure to trichloroethylene may result in headaches, fatigue, nausea, vomiting, mental confusion, and visual disturbances. Addiction to a craving for futher exposure sometimes develops.

On repeated or prolonged contact with skin, trichloroethylene exposure may result in the development of dermatitis. Frequent contact of the solvent with the skin will dry it by removal of the skin oils, making it rough and subject to cracking. This can lead to secondary infection.

On oral ingestion of trichloroethylene the initial symptoms are those of irritation of the gastrointestinal tract, such as nausea and vomiting and diarrhea. These may be followed by the development of anesthesia just as it develops after inhalation of the compound.

## REFERENCES


1. Stüber, K.: Gesundheitschädigungen bei der gewerblichen Verwendung des Trichlorethylens und die Möglichkeiten ihrer Verhütung, Arch. Gewerbepath. u. Gewerbehyg. 2:398, 1931.
2. Manufacturing Chemists' association, Inc.: Chemical Safety Data Sheet SD-14, Properties and Essential Information for Safe Handling and Use of Trichloroethylene, second revision, Washington, D.C., 1956.
3. Browning, E.: Toxicity of Industrial Organic Solvents, revised American edition, Chemical Publishing Co., New York, 1953, pp. 169-182.
4. Fairhall, L.T.: Industrial Toxicology, Ed. 2, The Williams & Wilkins Company, Baltimore, 1957, pp. 348-350.
5. von Oettingen, W.F.: The Halogenated Hydrocarbons — Toxicity and Potential Dangers, Public Health Service Publication No. 414, Washington, D.C., US Government Printing Office, 1955.
6. Barsoum, G.S., and Saad, K.: Relative Toxicity of Certain Chlorine Derivatives of the Aliphatic Series, Quart. J. Pharmacol. 7:205, 1934.
7. Lazarew, N.W.: Über die narkotische Wirkungskraft der Dämpfe der Chlorderivaten des Methans, des Äthans und des Äthylen, Arch. Exper. Path. u. Pharmakol. 141:19, 1929.
8. Carrieu, M.F.: Contribution à l'étude de l'intoxication par le trichlorure d'éthylène: Sa prophylaxie par le masque à l'huile, Rev. hyg. police sanit. 51:338, 1929.
9. Flury, F., and Zernik, F.: Schädliche Gase, Dämpfe, Nebel, Rauch und Staubarten, Berlin, Springer Verlag, 1931.
10. Lehmann, K.B.: Experimentelle Studien über den Einfluss technisch und hygienisch wichtiger Gase und Dämpfe auf den Organismus. Die gechlorten Koblenwasserstoffe der Fettreihe, Arch. Hyg., Berlin 74:1, 1911.
11. Taylor, H.: Experiments on the Physiological Properties of Trichloroethylene, J. Indust. Hyg. 18:175, 1936.
12. McCord, C.P.: Toxicity of Trichloroethylene, J.A.M.A. 99:409, 1932.
13. Genevois, L.: Sur les Propriétés physiologiques des solvents chlorés, Ann. hyg. pub. Paris 14: 139, 1936.
14. Barrett, H.M.; Maclean, D.L., and Cunningham, J.G.: Comparison of the Toxicity of Carbon Tetrachloride and Trichlorethylene, J. Indust. Hyg. 20:360, 1938.
15. Herzberg, M.: Histology of Tissues Taken from Animals Killed by Prolonged Administration of Concentrated Vapors of Trichlorethylene Cur. Res. Anesth. 13:203, 1934.
16. Meyer, H.: Untersuchungen über die Giftwirkung des Trichlorathylens besonders auf das Auge, Klin. Monatsbl. Augenheilk. 82:309, 1929.
17. Adams, E.M.; Spencer, H.C.; Rowe, V.K.; McCollister, D.D., and Irish, D.D.: Vapor Toxicity of Trichloroethylene Determined by Experiments on Laboratory Animals, A.M.A. Arch. Indust. Hyg. 4:469, 1951.

18. Lehmann, K.B., and Schmidt-Kehl, L.: Die 13 wichtigsten Chlorkohlenwasserstofte der Fettreihe vom Standpunktder Gewerbhygiene, Arch. Hyg. Berlin 116:131, 1936.
19. Lehmann, K.B., and Flury, F.: Toxicology and Hygiene of Industrial Solvents, The Williams & Wilkins Company, Baltimore, 1943, pp. 177-184.
20. Seifter, J.: Liver Injury in Dogs Exposed to Trichloroethylene, J. Indust. Hyg. & Toxicol. 26:250, 1944.
21. Mogilevskaya, S.Y.: Improving working conditions in degreasing metals with trichloroethylene, Gig. i sanit. 10: No. 12, p. 25, 1945.
22. Quadland, H.P.: Petroleum Solvents and Trichloroethylene — Part 3 of the Literature Study of Reports of Occupational Diseases Attributed to Volatile Solvents, Indust. Med. 13:45, 1944.
23. Striker, C.; Goldblatt, S.; Warn, I.S. and Jackson, D.E.: Clinical Experience with the Use of Trichlorethylene in the Production of Over 300 Analgesics and Anesthesias, Cur. Res. Anesth. 14: 68, 1935.
24. Kleinfeld, M., and Tabershaw, I.R.: Trichloroethylene Toxicity: Report of 5 Fatal Cases, A.M.A. Arch. Indust. Hyg. 10:134, 1954.
25. Geiger, A.J.: Cardiac Dysrhythmia and Syncope from Therapeutic Inhalation of Chlorinated Hydrocarbon, J.A.M.A. 123:141, 1943.
26. Berstine, M.L.: Cardiac Arrest Occurring Under Trichloroethylene Analgesia: A Case with recovery, A.M.A. Arch. Surg. 68:262, 1954.
27. Waters, R.M.; Orth, O.S., and Gillespie, N.A.: Trichloroethylene Anesthesia and Cardiac Rhythm, Anesthesiology 4:1, 1943.
28. Bell, A.: Death from Trichloroethylene in a Drycleaning Establishment, New Zealand M.J. 50: 119, 1951.
29. Chenoweth, M.B.: Ventricular Fibrillation Induced by Hydrocarbons and Epinephrine, J. Indust. Hyg. & Toxicol. 28:151, 1946.
30. Barrett, H. M., and Johnston, J.H.: The Fate of Trichlorethylene in the Organism, J. Biol. Chem. 127:765, 1939.
31. Barrett, H.M.; Cunningham, J.G., and Johnston, J.H.: A study of the Fate in the Organism of Some Chlorinated Hydrocarbons, J. Indust. Hyg. & Toxicol. 21:479, 1939.
32. Powell, J.F.: Trichlorethylene: Absorption, Elimination and Metabolism, Brit. J. Indust. Med. 2:142, 1945.
33. Ahlmark, A., and Forssman, S.: Evaluating Trichloroethylene Exposures by Urinalyses for Trichloroacetic Acid, A.M.A. Arch Indust. Hyg. 3:386, 1951.
34. Grandjean, E.; Munchinger, R.; Turrian, V.; Haas, P.A.; Knoepfel, H.K., and Rosenmund, H.: Investigations into the Effects of Exposure to Trichloroethylene in Mechanical Engineering, Brit. J. Indust. Med. 12:131, 1955.
35. Fabre, R., and Truhaut, R.: Contribution à l'étude de la Toxicologie du trichloréthylène, Brit. J. Indust. Med., 9:39, 1952.
36. Forssman, S.; Owe-Larsson, A., and Skog, E.: Metabolism of Trichloroethylene in the Organism, Arch. Gewerbepath. u. Gewerbehyg. 13:619, 1955; abstracted, A.M.A. Arch. Indust. Health 14: 194, 1956.
37. Stockman, S.: Cases of Poisoning in Cattle by Feeding on Meal from Soybean After Extraction of the Oil, J. Comp. Path & Therap. 29:95, 1916.
38. McKinney, L.L.; Uhing, E.H.; White, J.L., and Picken, J.C., Jr.: Autoxidation Products of Trichloroethylene, J. Agric. & Food Chem. 3:413, 1955.
39. Pritchard, W.R.; Hammer, R.; Sautter, J.H., and Schultze, M.O.: Susceptibility of the Horse to the Toxic Factor in Trichloroethylene-Extracted Soybean Oil Meal, Am. J. Veterinary Res. 17:441, 1956.
40. Pritchard, W.R.; Perman, V.; Mattson, W.E.; Sautter, J.H., and Schultze, M.O.: The Effects of Feeding Trichloroethylene-Extracted Soybean Oil Meal to Sheep, Am. J. Veterinary Res. 17:446, 1956.
41. Bleyer, B., and Meyer, K.: Zur Frage der Ursachen der Dürener Rinderkrankheit, Fortschr. Landwirtschaft 2:173, 1927.
42. McKinney, L.L.; Weakly, F.B.; Eldrige, A.C.; Campbell, R.E.; Cowan, J.C.; Picken, J.C. Jr., and Biester, H.E.: S-(Dichlorovinyl)-L-Cysteine: An Agent Causing Fatal Aplastic Anemia in Calves, J. Am. Chem. Soc. 79:3932, 1957.
43. American Conference of Governmental Industrial Hygienists: Threshold Limit Values for 1958, A.M.A. Arch. Indust. Health 18:178, 1958.
44. American Industrial Hygiene Association. Hygienic Guides Committee: Trichloroethylene, Am. Indust. Hyg. A. Quart. 17:95, 1956.
45. Smyth, H.F.: Hygienic Standards for Daily Inhalation, Am. Indust. Hyg. A. Quart. 17:178, 1956.
46. Stokinger, H.E.: Trichloroethylene Exposure Problem, A.M.A. Arch. Indust. Health 14:210, 1956.
47. Imperial Chemical Industries, Ltd.: Toxic Concentrations of Various Gases, Dusts, Fumes and Metals in the Atmosphere, Indust. Hyg. Digest 22: No. 10 (Oct.) 1958.
48. Ahlmark, A., and Friberg, L.: Should the Maximal Allowable Concentration of Trichloroethylene and Similar Substances be Reduced ? Sartyck u. Nord. Hyg. Tidskr. 36:165, 1955.

## NOTICE

In order to have a complete set of the Occupational Health Review, we require Volume 4, No. 1, October, 1952.

If any of our readers have this volume and do not wish to keep it, it would be appreciated if they would mail it to: The Editor,
Occupational Health Division,
Department of National Health and Welfare,
45 Spencer Street,
Ottawa, Ontario.

No postage will be required if the envelope is marked "O.H.M.S.".

OTTAWA
Queen's Printer and Controller of Stationery
1960

# OCCUPATIONAL HEALTH *Review*

Published by the Occupational Health Division department of National Health and Welfare, Ottawa with the co-operation of the department's Information Services Division and under the authority of the Minister, the Hon. J. Waldo Monteith.

A QUARTERLY PUBLICATION